Dissertation on

A CLINICAL STUDY ON
CENTRAL SEROUS CHORIORETINOPATHY

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

BRANCH - III

REGIONAL INSTITUTE OF OPHTHALMOLOGY

MADRAS MEDICAL COLLEGE

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DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2012

CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL STUDY ON CENTRAL SEROUS CHORIORETINOPATHY"** is a bonafide record of the research work done by **Dr.N.VIMALKUMAR.,** Post graduate in Regional Institute of Ophthalmology, Madras Medical College and Research Institute, Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2009-2012.

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ACKNOWLEDGEMENT

I express my sincere thanks and gratitude to **Prof. Dr.V.KANAGASABAI M.D.,Ph.D.,** Dean, Madras Medical College for permitting me to conduct this study.

I have great pleasure in thanking **Prof**. **Dr. K. VASANTHA, M.S., FRCS.,** Director and Superintendent RIO – GOH, Madras Medical College, Chennai, for her valuable advice in preparing this dissertation.

I express my profound gratitude to **Prof**. **Dr.R.RAVIKUMAR M.S DO.,** my unit chief and my guide for his valuable guidance and constant support at every stage throughout the period of this study.

I am very grateful to my unit assistants **Dr. ASHOK KUMAR M.S., Dr. R. PADMAPRIYA M.S.,** and **Dr. A. PALANIRAJ M.S.,** for rendering their valuable advice and guidance for the study.

I wish to express my sincere thanks to all the professors, assistant professors and all my colleagues who had helped me in bringing out this study.

Finally, I am indebted to all the patients for their sincere co-operation for the completion of this study.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled, **"THE CLINICAL STUDY ON CENTRAL SEROUS CHORIORETINOPATHY,"** is a bonafide and genuine research work conducted by me under the guidance of Prof. Dr.R.Ravikumar M.S.,D.O., Professor, Department of Uvea/Retina services, Regional institute of ophthalmology, Government Ophthalmic hospital, Chennai-600008.

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PLACE

CONTENTS

PART ONE

ABBREVIATIONS

CSCR-CENTRAL SEROUS CHORIORETINOPATHY

PED-PIGMENT EPITHELIAL DETACHMENT

FAZ-FOVEAL AVASCULAR ZONE

FFA-FUNDUS FLUORESCEIN ANGIOGRAPHY

ICG-INDOCYANINE GREEN ANGIOGRAPHY

OCT-OPTICAL COHERENCE TOMOGRAPHY

CNVM-CHOROIDAL NEOVASCULAR MEMBRANE

RPE-RETINAL PIGMENT EPITHELIUM

IS-OS-INNER SEGMENT-OUTER SEGMENT JUNCTION

NSAIDS-NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

INTRODUCTION

CENTRAL SEROUS CHORIORETINOPATHY is a disease characterized by spontaneous serous macular detachment of unknown etiology, usually self-limiting but often recurs.

It was first described by VonGraefe in 1866 and termed it “central recurrent retinitis”, “central angiopathic retinopathy”.[1]

Kitahara in 1936 described it as chorioretinitis centralis serosa.

Walsh and solan described it as Idiopathic flat detachment of macula.

In 1955 Bennet coined the term central serous retinopathy.

Klein and Maumenee used fundus fluorescein angiography to observe leaks at the level of retinal pigment epithelium.

In 1967 Gass provided the pathogenesis and labelled it as “Idiopathic central serous choroidopathy”.

ANATOMY OF MACULA

Macula is that portion of the posterior pole with minimum two layers of nuclei in the ganglion cell layer and contains xanthophyll pigment. There is no anatomical landmark to define this zone on clinical examination or on morphological basis. It is approximately a circle with a radius of 2.75mm centered at fovea (5.5mm in diameter).[3]

Fovea centralis is the depression in the inner retinal surface in the centre of the macula and is 1.5mm in diameter.

Foveola is 0.35mm in diameter. It is situated 4mm temporal and 0.8mm inferior to the optic nerve head. Rod-Cone ratio is about 1:2 in this region.

A small depression in the centre of foveola is called umbo.

Parafoveal zone is an area measuring 0.5 mm surrounding the fovea. Rod-Cone ratio is about 1:1. Perifoveal area is 1.5mm wide zone surrounding parafoveal area.[3]

Retinal pigment epithelial cells at the fovea are taller, thinner, contain more and large pigment granules than elsewhere in the fundus and this makes the macula appear darker than the rest of fundus.

Blood supply of Macula

The macula region get its blood supply by small twigs from the superior and inferior branches of the central retinal artery. In 20% of individuals cilioretinal artery, a branch from the ciliary system of vessels supply the macula. Capillaries are arranged as three layered in the macula, and they are reduced to single layer in the perifoveal area and in centre is the capillary free zone of 400-600 µm in diameter.[3]

BLOOD- RETINAL BARRIER

Outer Blood Retinal Barrier

This is formed by the tight junctions (Zonulae occludens and Zonulae adherens) of retinal pigment epithelial cells.

Inner Blood-Retinal barrier

The endothelial cells of retinal capillaries bound together, about the lumen by intercellular junctions of zonula occludens type and forms the inner Blood-Retinal barrier.[3]

RPE and CHOROID

Retinal pigment epithelial cells are densely adherent to the underlying Bruch's membrane of choroid. The oncotic pressure, exerted by the intravascular proteins of choriocapillaries and the intracellular pumping mechanism within the RPE maintains the dehydrated state of the sub-retinal space.

The Choroid is supplied by the ciliary system that is concentrated in the macula and peripapillary region. They form rich anastomotic network and there is a rapid transformation from arterioles to capillaries. In the macula, there is a lobular pattern of arrangement, which facilitates rapid blood flow.[3]

MICROANATOMY OF MACULA

Retina, at the macula consists of 3 types of cells and their synapses arranged from without inwards in the following layers,

- Retinal pigment epithelium
- Layer of Rods and Cones
- External limiting membrane
- Outer nuclear layer
- Outer plexiform layer
- Inner nuclear layer
- Inner plexiform layer
- Ganglion cell layer (multilayered in comparison to rest of retina)
- Nerve fibre layer
- Internal limiting membrane.[3]

In Central Serous Chorioretinopathy there is a split between the RPE and rest of the layers of neurosensory retina at the macula.

FOVEA CENTRALIS

This region is predominated by cones and their axons are arranged obliquely, forming the henle's layer. It contains 10% of the cone population in the whole retina.

FOVEOLA

This region of retina contains cones and their nuclei covered by thin internal limiting membrane. Remaining retinal layers are absent in the macula.[3]

EVALUATION OF MACULAR DISEASES

SLIT LAMP BIOMICROSCOPY

It ulilizes high power convex lenses to obtain wide field of view of the fundus which is vertically inverted and laterally reversed, It provides high magnification with stereopsis to detect macular disease.[4]

AMSLER GRID

It evaluates the 20° of visual field centred on fixation, and hence useful in screening and monitoring the macular disease. There are 7 charts. Chart 1is most commonly used. This chart consists of white grid on black background, with 400 smaller 5mm squares, each square subtends an angle of 1° when viewed at 33cm. Each eye is checked individually, with the chart held at 33 cm, priorly patients should be corrected for the presbyopia. Patients are asked to maintain fixation on the central dot and comment on the four corners, of the sides, any missing areas on chart and wavy lines.[4]

FUNDUS FLUORESCEIN ANGIOGRAPHY

Fluorescence is the property of certain molecules to absorb light of shorter wavelength and emit light of longer wavelength. This is the principle in fundus fluorescein angiography and is extremely valuable in evaluation of macular diseases.[4,5]

Sodium fluorescein, an orange water soluble dye, about 3ml of 25% is injected intravenously, 85% of it is bound to plasma proteins and remains intravascular, passage of dye through the retinal and choroid circulation is studied through photographic surveillance.[4,5]

PHASES IN FFA

- Choroidal phase
- Arterial phase
- Arteriovenous phase
- Venous phase
- Recirculation phase.

Causes of Hyperfluorescence

Autofluorescence, Pseudofluorescence, Window defect, Pooling, leakage and staining.

Causes of Hypofluorescence

Masking of Retinal fluorescence, masking of choroidal fluorescence, Filling defects.[4,5]

INDOCYANINE GREEN ANGIOGRAPHY

Masking effect of RPE prevents the delineation of choroidal vasculature in FFA. ICG utilizes the near infrared light for deep penetration. ICG is 98% protein bound and does not leak through the fenestrations of choriocapillaries and remains within the choroidal vasculature and helps to study the choroidal disease.

A dose of 25-50mg in 1-2ml is injected through the antecubital vein.

Phases of choroidal fluorescence

- Early phase (upto 60 sec)
  - Shows choroidal arteries and early perfusion of watershed zone.

- Early mid-phase (1-3mins)
    - Shows Choroidal veins and Retinal vessels.

- Late mid-phase (3-15mins)
    - Shows fading of choroidal vessels and retinal vessels still visible.

- Late phase (15-45mins)
    - Shows hypofluorescence of choroidal vessels and gradual fading of diffuse hyperfluorescence.

Causes of hyperfluorescence

- Window defect
- Abnormal retinal or choroidal vessels
- Leakage

Causes of hypofluorescence

- Blockage
- Filling defects

ICG is indicated in macular diseases like CNVM, CSCR, in Polypoidal choroidal vasculopathy, PED, posterior uveitis, choroidal tumour and so on.[4]

OPTICAL COHERENCE TOMOGRAPHY

OCT is a noninvasive, noncontact imaging system provides high resolution cross-sectional images of the retina, optic nerve head and the vitreous.

Principle

OCT is based on imaging of reflected light (near infrared light), analogous to B-Scan, the only difference is that OCT uses the principle of low coherence interferometry measures the optical rather than acoustic or radio wave reflectivity.

OCT is indicated to differentiate the lamellar and full thickness macular hole, to determine the treatment option in CNVM, monitoring the course of CSCR and locate the area of leak, retinal thickness map and so on.[6]

HIGH REFLECTIVITY

- Nerve Fibre Layer (Normal)
- RPE-Choriocapillaries (Normal)
- Pigment accumulation
- Naevus
- Neovascularization
- RPE hypertrophy

MEDIUM REFLECTIVITY

- Plexiform Layer (Normal)

LOW REFLECTIVITY

- Nuclear Layer (Normal)
- Photoreceptors (Normal)
- Retinal Edema
- Cystoid Edema
- Cavity
- Cyst
- Pigment Epithelial Detachment
- Serous Retinal Detachment[10]

EPIDEMIOLOGY OF CENTRAL SEROUS CHORIORETINOPATHY

Central serous chorioretinopathy is characterized by an idiopathic circumscribed serous retinal detachment usually confined to central macula, caused by the leakage of fluid through retinal pigment epithelium.

CSCR is most common in middle age adults between 20-45 years, but cases as old as 50yrs and as young as 7yrs are reported.[8] CSCR affects men more frequently than women (male to female ratio 6:1).[9]

Caucasians are affected frequently and Afro- americans the least. Type A personality, patients on long term steroids in any form (topical, oral, inhalational or injectable) systemic steroids in organ transplant patients, rarely following PST and intravitreal steroids, pregnancy, alcohol, antibiotics, untreated hypertension.[10]

PATHOPHYSIOLOGY OF CSCR

Various theories have been put forward and yet to evolve, as we understand the disease better as investigation advances.

FFA shows serous detachment which is due to the accumulation of fluid from the choroid through a precisely located defect in the RPE.

ICG angiography gives a new graphic abnormality of choroidal circulation in patients with central serous chorioretinopathy.

Raised Sympathomimetics levels in circulation

↓

Increased venous congestion of choroidal vasculature

↓

Multifocal areas of hyperpermeability in choroidal circulation

and increased tissue hydrostatic pressure within the vasculature leads

to pigment epithelial detachment

↓

Disruption of retinal pigment epithelial barrier

↓

Abnormal egress of fluid under the retina leading to CSCR.[11,12]

HISTOPATHOLOGY OF CSCR

Shows focal areas of degenerated RPE with adjacent damaged choriocapillary endothelial cells.

The presence of subretinal fibrin indicates alteration in the permeability of choriocapillaries permitting the molecules as large as fibrinogen to leak.[13]

CLINICAL FEATURES OF CSCR

SYMPTOMS

- Minimal blurring of vision
- Metamorphopsia
- Dyschromatopsia
- Hypermetropisation
- Central scotoma
- Loss of contrast sensitivity

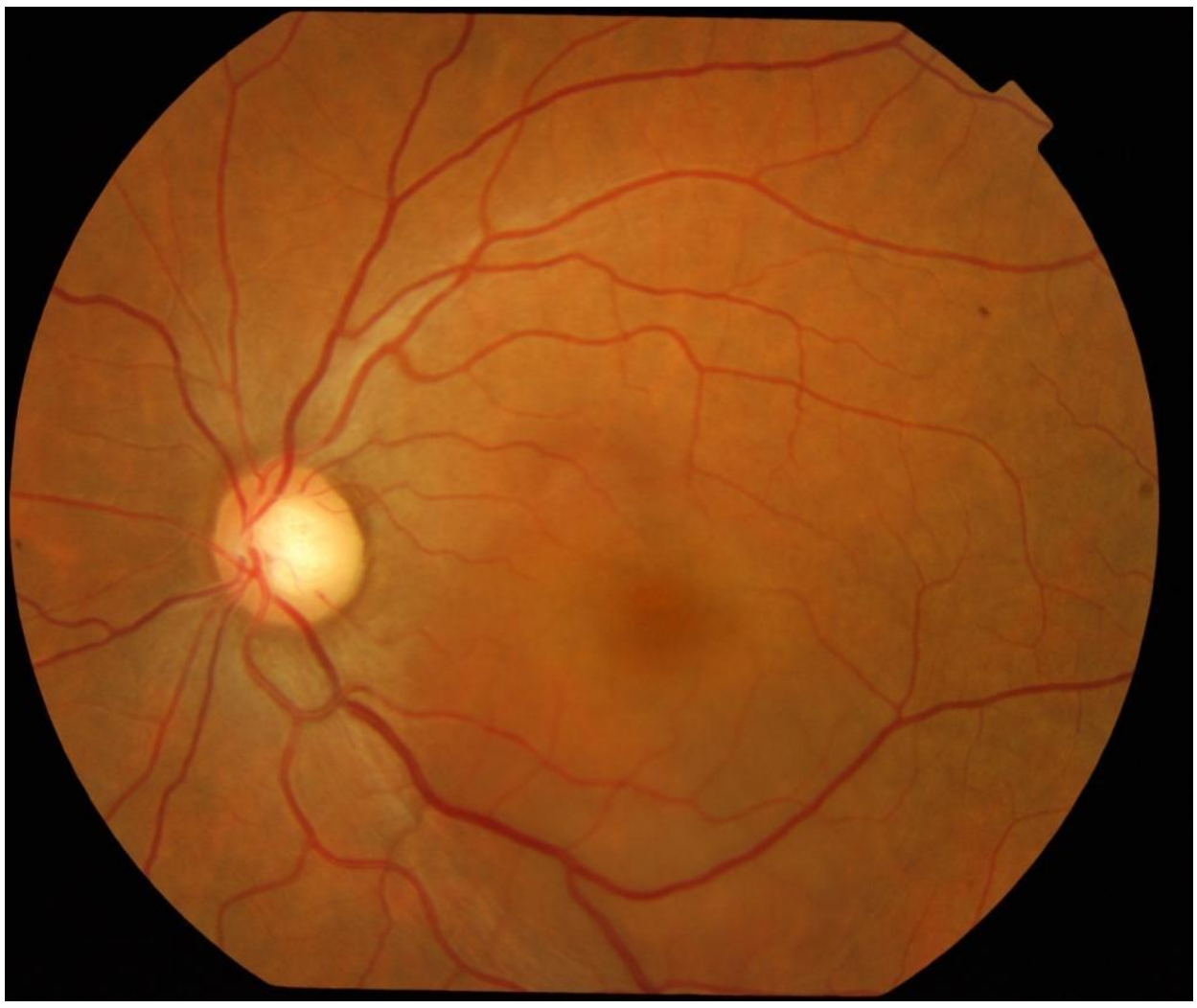
SIGNS

Fundus Examination shows localized detachment of retina at the macula appearing as delineated transparent blister at the posterior pole.(FIG-1)

Pigment epithelial detachment can also be seen.[14]Subretinal deposits are occasionally seen, these occur in four forms which includes fibrin, lipid, macrophages and outer photoreceptor segments.Hourglass pattern of RPE abnormalities seen in chronis CSCR.(FIG-2)

Subretinal lipid and macrophages occur in chronic CSCR patients.[15]Multifocal serous detachment rarely reported in some patients (FIG-3,4)

FIGURE-1-SEROUS DETACHMENT IN MACULA



FIGUREP-2-CSR WITH HOURGLASS PATTERN OF RPE DEFECTS

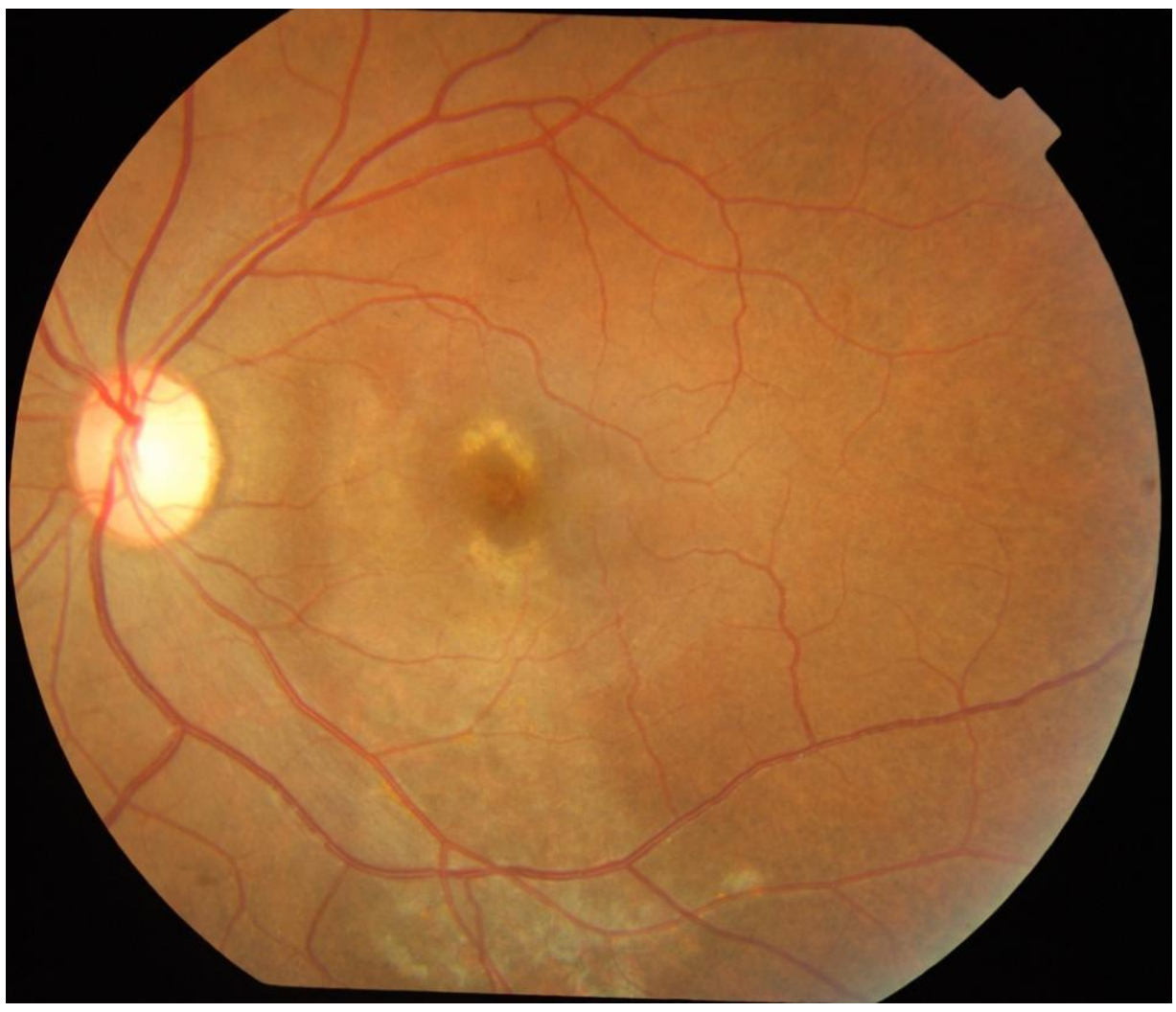


FIGURE-3 MULTIFOCAL DETACHMENT

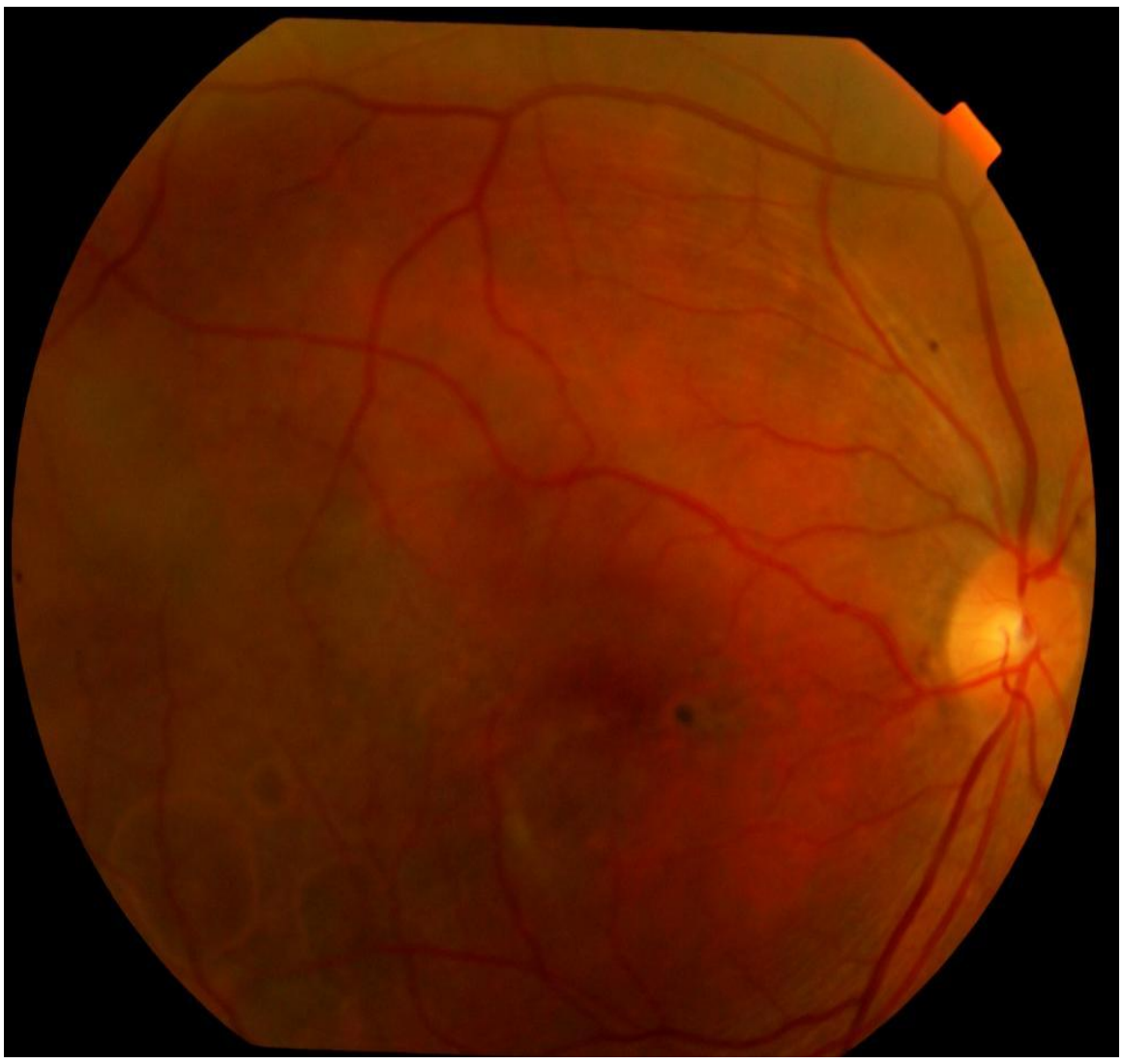
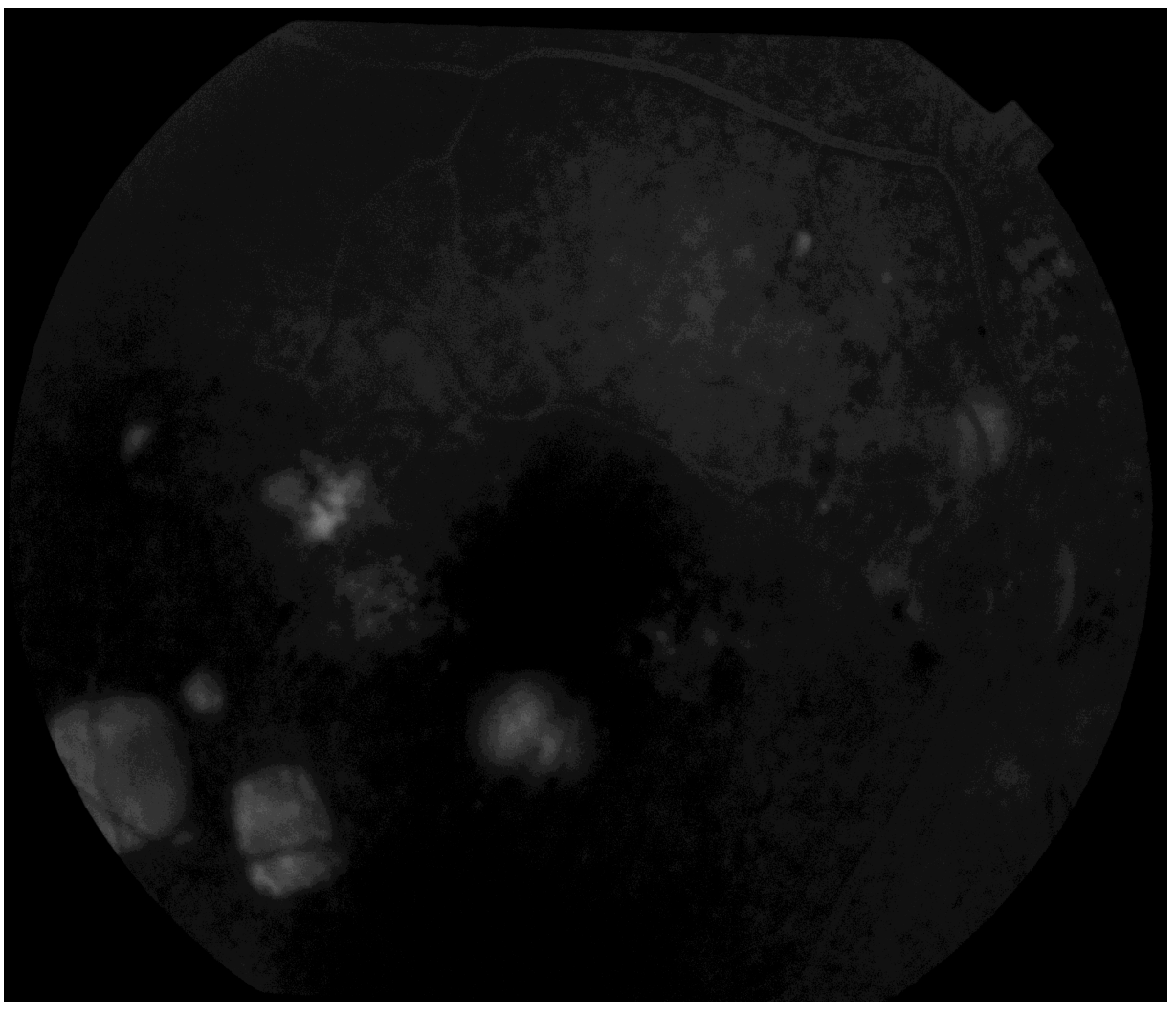


FIGURE-4 MULTIFOCAL DETACHMENT IN FFA



IMAGING MODALITIES IN CSCR

Fundus Fluorescein Angiography

The typical, most common dye leakage pattern in CSCR is "Ink blot pattern"(FIG-5) present in 90% of cases.[16,17]

Others include "Smoke stack pattern"(FIG-6,7,8) its also termed as mushroom or umbrella pattern.

The reason for Smoke stack pattern is due to convection current and differences in protein gradient of fluid in the subretinal space.[16]

The dye spreads in subretinal space in late phase of FFA and shows pooling of the dye in the detached area.

In acute cases, increased autofluorescence noted at the leakage site and in the areas of retinal detachment. This was attributed to increased metabolic activity of RPE.

In chronic cases, autofluorescence is due to subretinal granular deposits.[18,19]

FIGURE-5 INK BLOT PATTERN IN LATE PHASE

FIGURE-6 SMOKE STACK PATTERN –EARLY PHASE

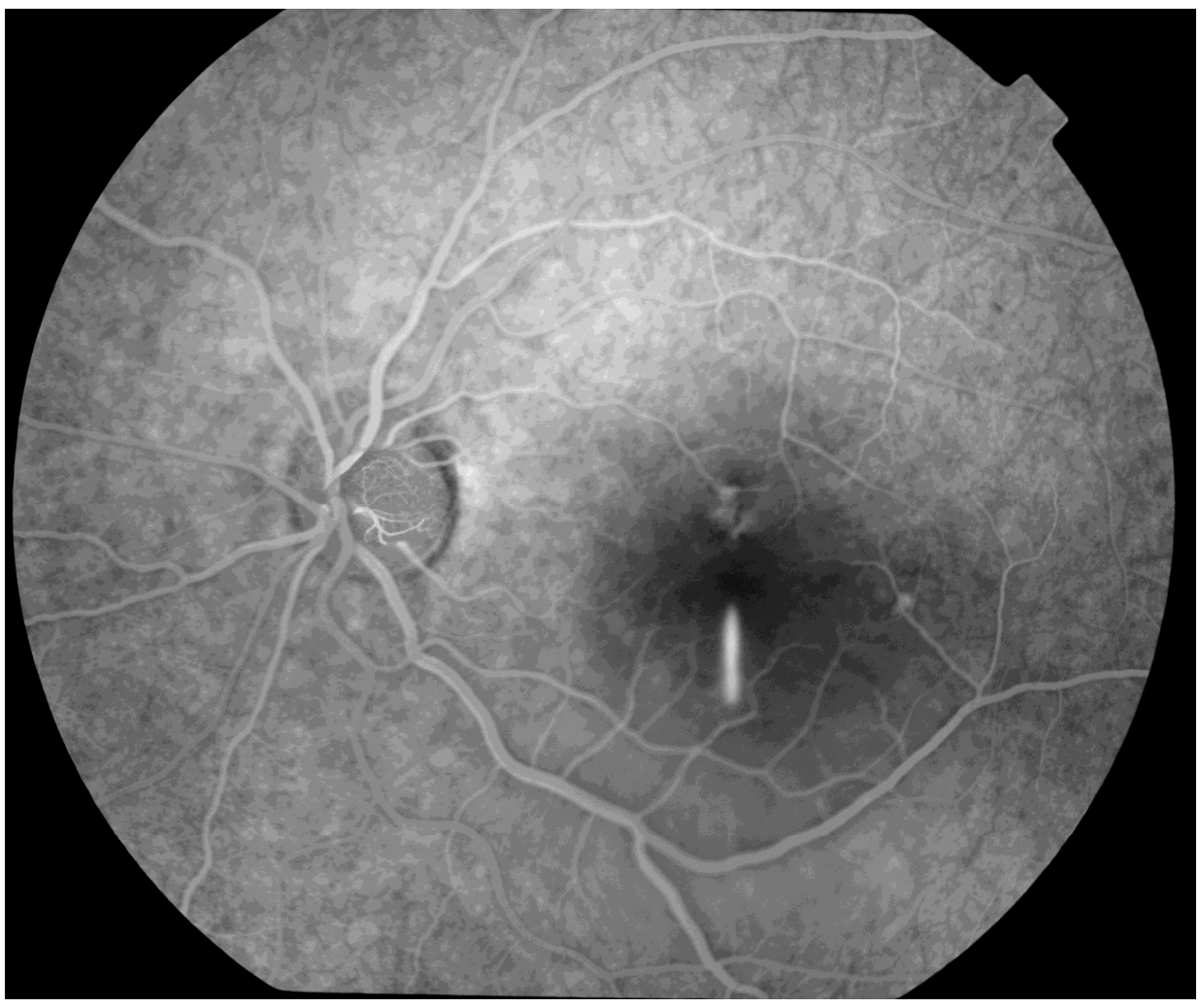
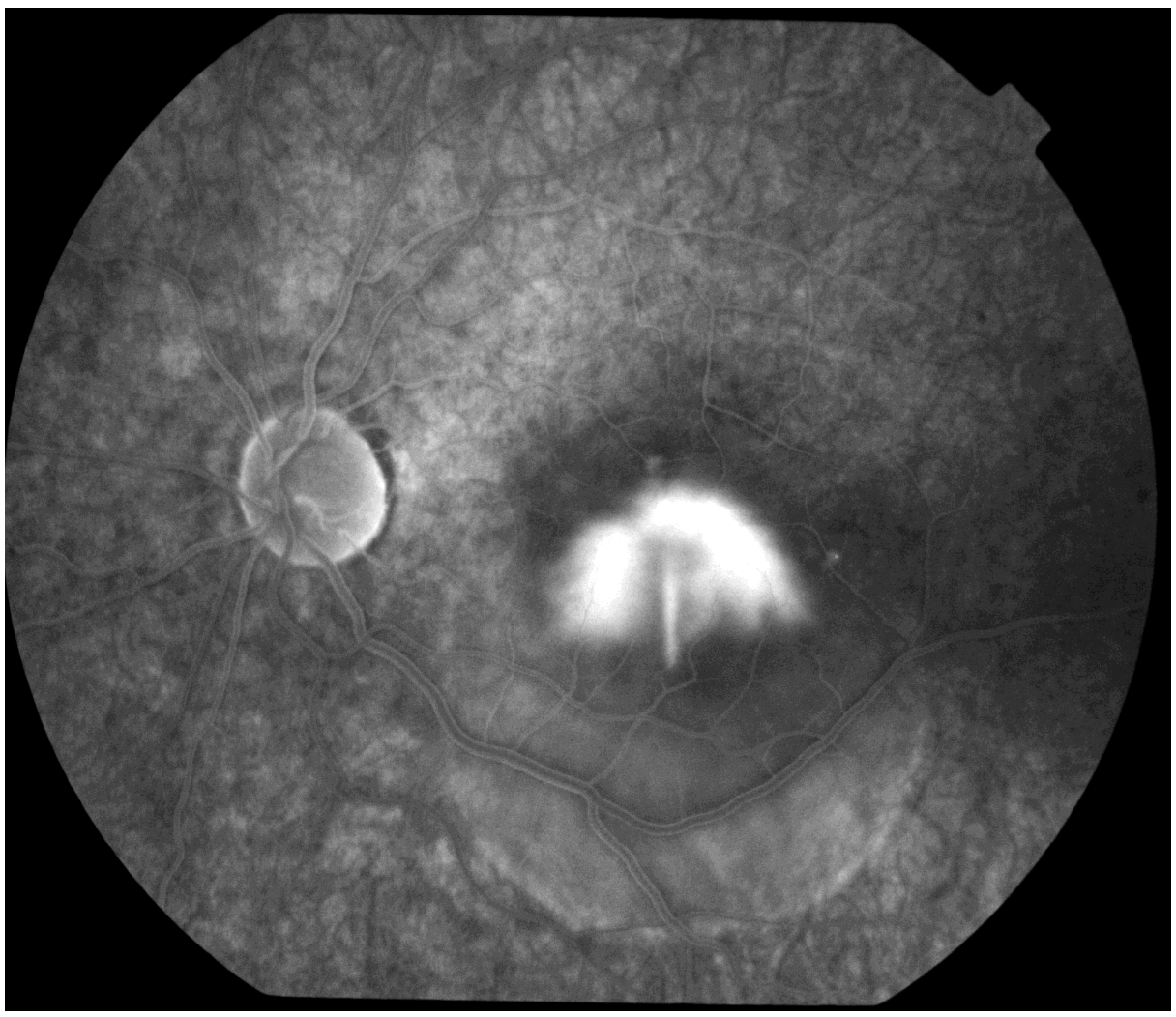


FIGURE-7 SMOKE STACK PATTERN IN MID-PHASE

FIGURE-8 SMOKE STACK PATTERN IN LATE-PHASE



Indocyanine Green Angiography

ICG demonstrates hyperpermeability of the choriodal vasculature, best seen in mid-phase of the angiogram.

In the late phase, leaked dye appears to disperse, producing a characteristic appearance of hyperfluorescent patches in the choroid with silhouetting of the large choroidal vessels.

Young patients have PED as a forme fruste of CSCR where the underlying choroidal hyperpermeability may cause elevation of RPE without causing a breakthrough leak.[20,8]

Optical Coherence Tomography

OCT is effective in diagnosing and quantifying the serous detachment of retina.

OCT shows bullae of subretinal fluid with near normal neuroretinal architecture overlying it(FIG-9).

Retinal atrophy may be noted in some patients.Pigment epithelial detachment is noted in some patients.(FIG-10).

Spectral domain OCT additionally demonstrates elongation of photoreceptor outer segment and IS-OS junction distance, hyperreflectivity of outer nuclear and inner plexifom layer and choroidal thickening in CSCR.[21.22]

FIGURE-9SEROUS DETACHMENT OF MACULA

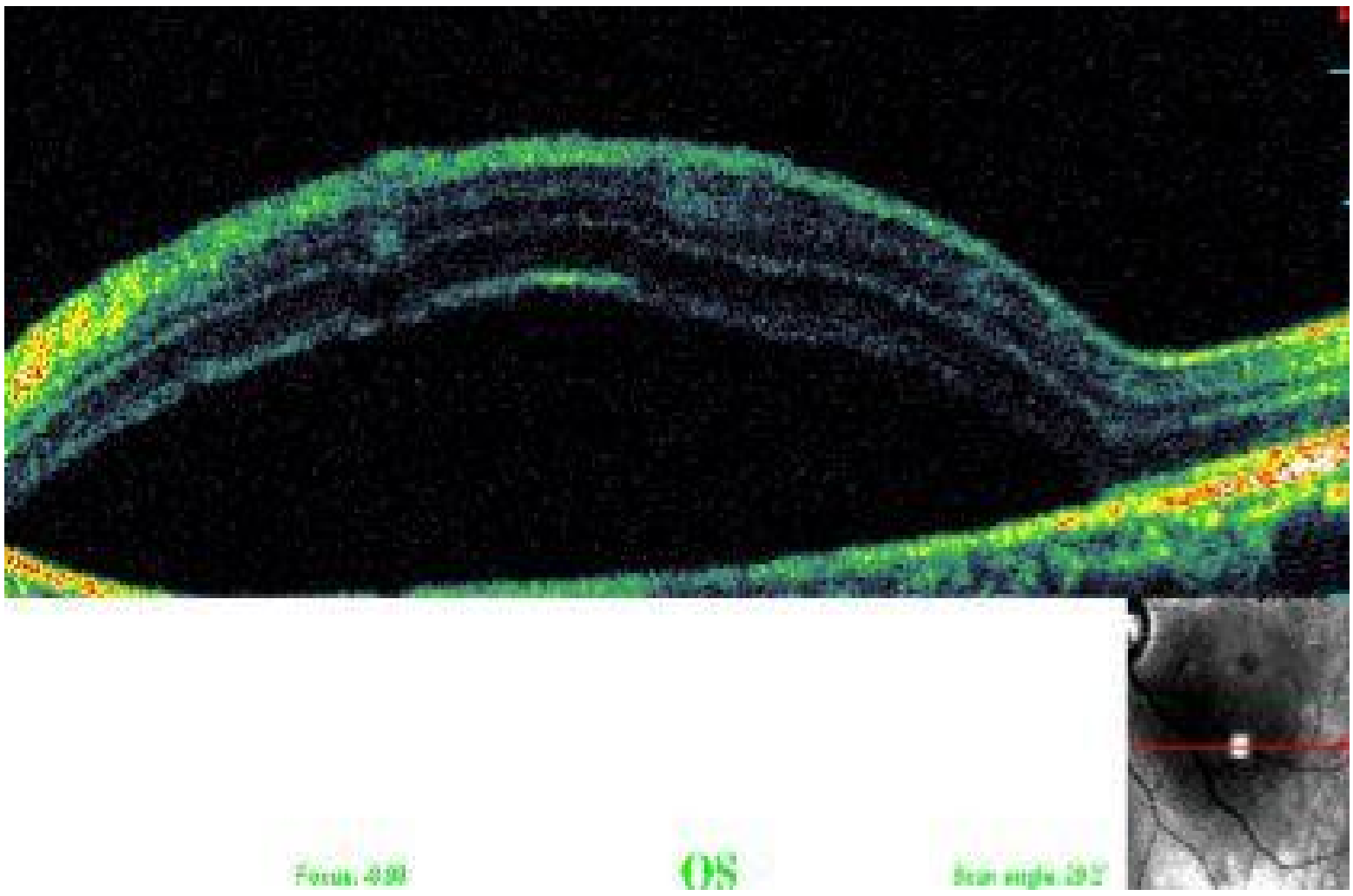
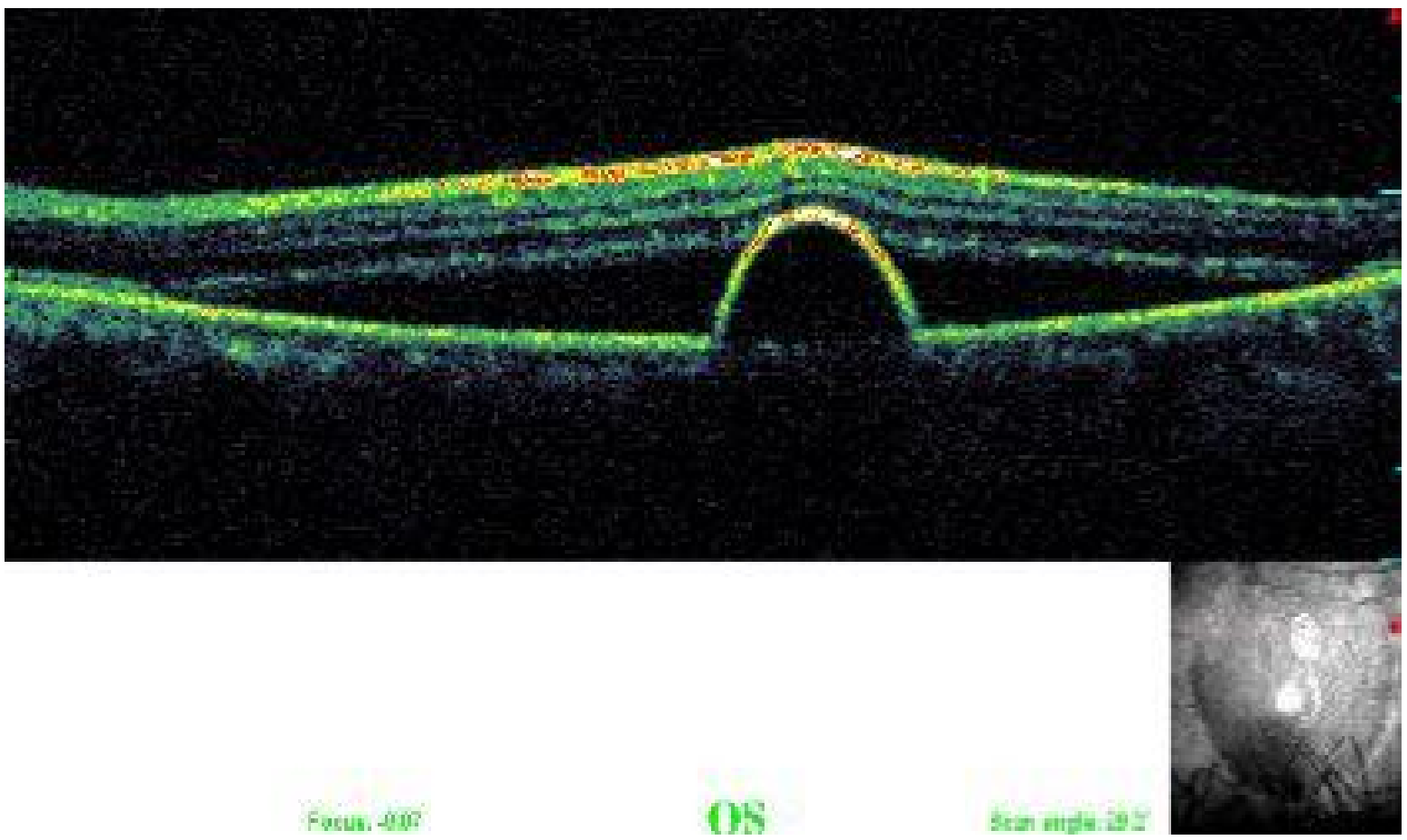


FIGURE-10 SEROUS DETACHMENT WITH PED



DIFFERNTIAL DIAGNOSIS OF CSCR

- Harada's disease
- Posterior scleritis
- Sympathetic ophthalmitis
- Optic disc pit
- Choroidal neovascular membrane
- Choroidal melanoma
- Choroidal hemangioma
- Benign reactive hyperplasia of the choroid
- Uveal effusion syndrome
- Polypoidal choroidal vasculopathy
- Malignant hypertension
- Toxemia of pregnancy.[15]

TREATMENT MODALITIES IN CSCR

CSCR is self resolving in most cases with visual recovery of 20/25 or better.

Patients advised to avoid the precipitating causes of the disease. Systemic steroids in any form of administration (oral, inhalational, topical, ointments, etc)[23,24]

Majority of patients with CSCR were associated with raised serum cortisol level.[25]

Factors Determining The Treatment Modality

- Visual acuity
- Recurrences
- Visual needs

Its generally recommended to observe a patient with CSCR for 3 months.

Treatment options include:

- Laser photocoagulation[26]
- Photodynamic therapy
- Transpupillary thermotherapy
- Medical treatment
- Meditation and yoga

Indications for early laser photocoagulation:

- Multifocal CSCR
- Bullous sensory detachment
- Visual loss in fellow eye due to CSCR.
- Recurrent CSCR
- Diffuse RPE decompensation
- Subretinal deposits of fibrin and lipid
- Patient on steroids where therapy cannot be withdrawn
- All patients with CSCR where leak is not threatening the fovea[30,31]

Laser Photocoagulation

Watze et al reported that laser photocoagulation hastened the resolution of CSCR from 25 wks to 5wks.[28]

Most common laser used is

- Argon green
- Infrared laser
- Micropulsed diode laser

Technique

- Foci of leakage is localized with the help of FFA, 72 hrs before the procedure.
- Argon green Laser of spot size 50-100µm.
- Duration of 0.05-0.1sec
- Power of 70-80mW (to produce minimally visible burn of RPE)
- Laser is targeted at the site of leakage [27,28]
- Recently subthreshold diode laser can be tried for leaks within 500µm of the fovea.[32,33]

COMPLICATIONS OF LASER

- Scotoma
- Laser scars extend to fovea and cause visual detoriation
- Choroidal neovascularisation

The role of laser treatment is limited to hastening the resolution of symptomatic disease.

Photodynamic Therapy And Transpupillary Thermotherapy

- Indicated in chronic disease.[34,35]
- Scarring and collateral damage is the major drawback.

MEDICAL TREATMENT

- Beta blockers[36]
- Ketoconazole
- Multivitamin supplements
- Topical NSAIDS

All were tried with no conclusive benefits. Lifestyle modification like meditation, yoga can be tried, cessation of smoking should be stressed.

Prognosis

The prognosis for majority of the patients with typical CSCR is excellent with most patients having spontaneous resolution of macular detachment with return of visual acuity within 6 months, approximately 5% may fail to recover 6/9 vision or better.

Some patients may develop permanent visual loss of 6/60 or less. Resolution of serous detachment demonstrates the evidence of irregular depigmentation.

The long term visual prognosis is good, approximately 20 -30% will have one or more recurrences.

PART TWO

AIM OF THE STUDY

- To study the FFA pattern and use of OCT (Optical coherence tomography) in CSCR.

- Study the effectiveness of early laser photocoagulation in CSCR patients with extrafoveal leakage in terms of visual acuity and central macular thickness and comparing with the control group.

- To study the epidemiology (incidence, recurrence, persistent cases) of CSCR.

- To study the association of CSCR with the use of steroids in any form (systemic, inhalers, ointment etc.,)

MATERIALS AND METHODS

This study was conducted in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Egmore, Chennai, from November 2009 to November 2011 for a period of 2 years. This is a single blind randomised prospective and retrospective inteventional clinical study.

INCLUSION CRITERIA

All new and review cases attending the Ophthal OPD with central serous retinopathy diagnosed clinically.

EXCLUSION CRITERIA

1. Patients with ocular infective and inflammatory condition,
2. Patients with diabetes,
3. Patients with collagen vascular disease

METHODOLOGY

- History,
- Visual acuity,
- Fields,
- Retinoscopy,

- Slit lamp examination,
- Amsler grid ,
- Fundus examination with90D & IDO
- FFA
- OCT

Patients diagnosed clinically as CENTRAL SEROUS CHORIORETINOPATHY were further analysed by taking, a brief history about the onset and duration of symptoms, history of recurrences (similar presentation in the same or the other eye) about the steroid usage in any form, patients were then subjected to a detailed systemic and ophthalmic evaluation.

OCT and FFA was done for all cases and leakage site identified. Patients with leakage in the foveal and juxtafoveal region(within 375 micrometer from centre of fovea) were isolated from the intervention.

Total number of patients reviewed with old records and diagnosed newly as CSCR were 50. Patients(Samples) with first incidence of CSCR and having FFA picture showing single site leakage which is 375µm away from the fovea were randomly divided into two subgroups by simple randomization.

Sample size was 30.

Subgroup1

About 15 patients from total of 30 were assigned randomly as subgroup1(study group) subjected to early laser photocoagulation.

Subgroup 2

Remaining 15 patients who were assigned as subgroup2 (control group) given placebo treatment.

Both the subgroups were subjected for visual acuity by snellen chart and central macular thickness documentation using OCT before and after early laser photocoagulation or placebo treatment as assigned previously.

During follow up visits at 4 weeks and 12 weeks visual acuity and central macular thickness post laser/post placebo treatment were noted.

In every follow up visits the following parameters were assessed

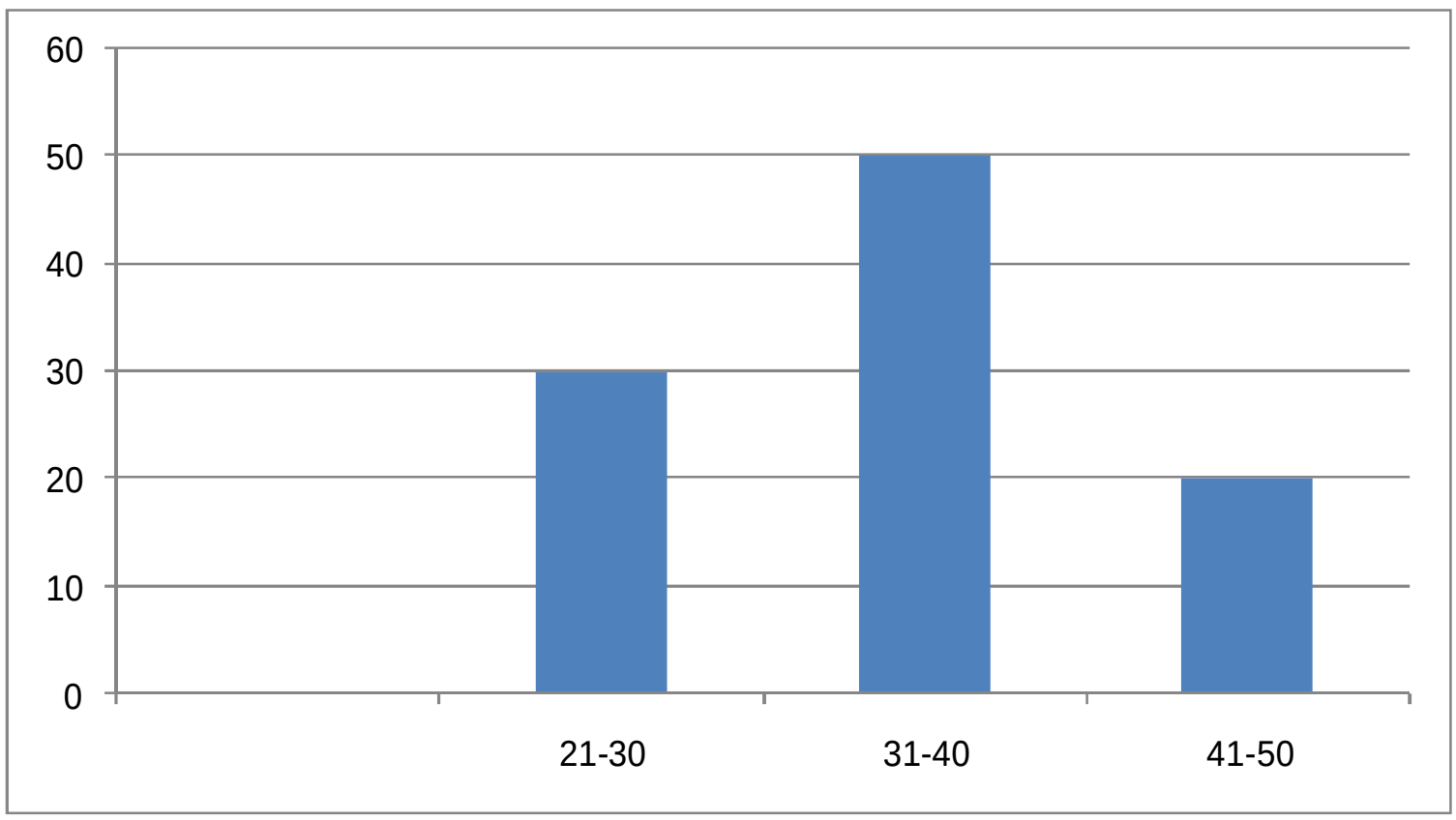
- Best corrected visual acuity
- Amsler grid
- Fundus examination
- OCT

OBSERVATION AND ANALYSIS

1. AGE DISTRIBUTION

Table - 1

AGE	NO	PERCENTAGE
21-30	15	30%
31-40	25	50%
41-50	10	20%

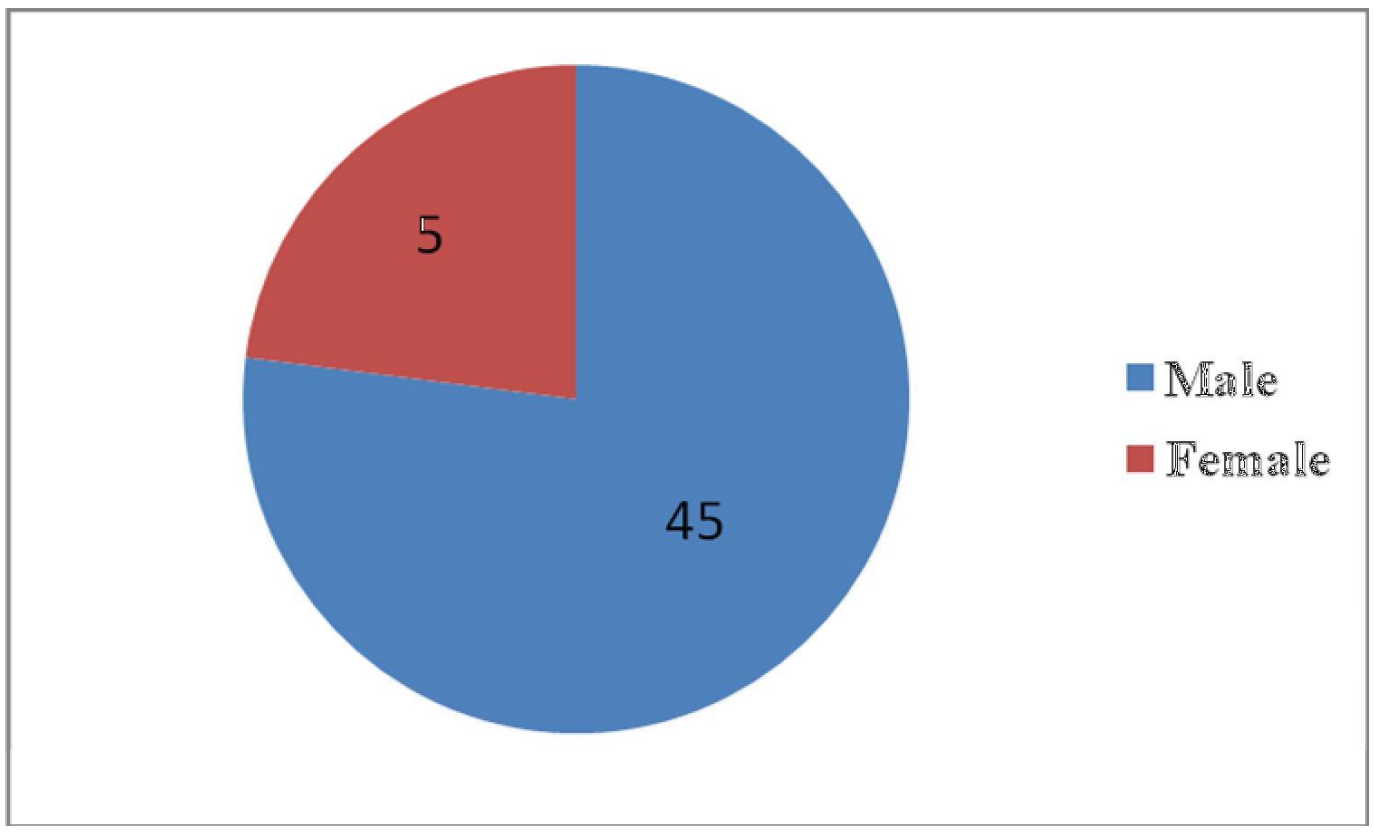


21-50 yrs is the predominant age group (50%) affected in our study. Gass et al in his study also showed predominant involvement between age groups 20 and 45 years.[11]

2. SEX DISTRIBUTION

Table - 2

Sex	No. of Cases	Percentage
Male	45	90%
Female	5	10%

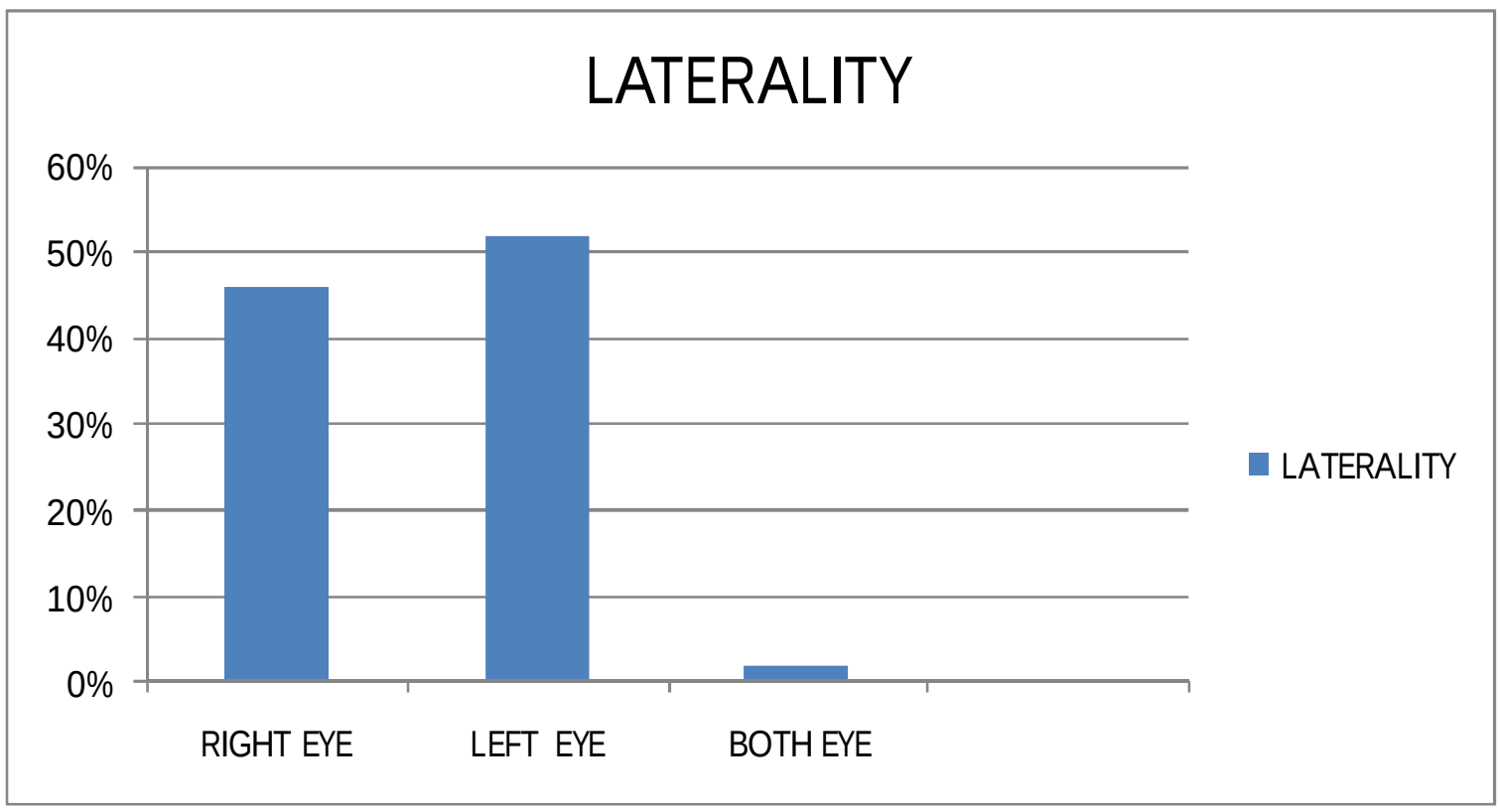


In our study males were predominantly affected (90%) compared to females.

3 LATERALITY

Table - 3

EYE	NO	PERCENTAGE
RIGHT	23	46%
LEFT	26	52%
BOTH	1	2%



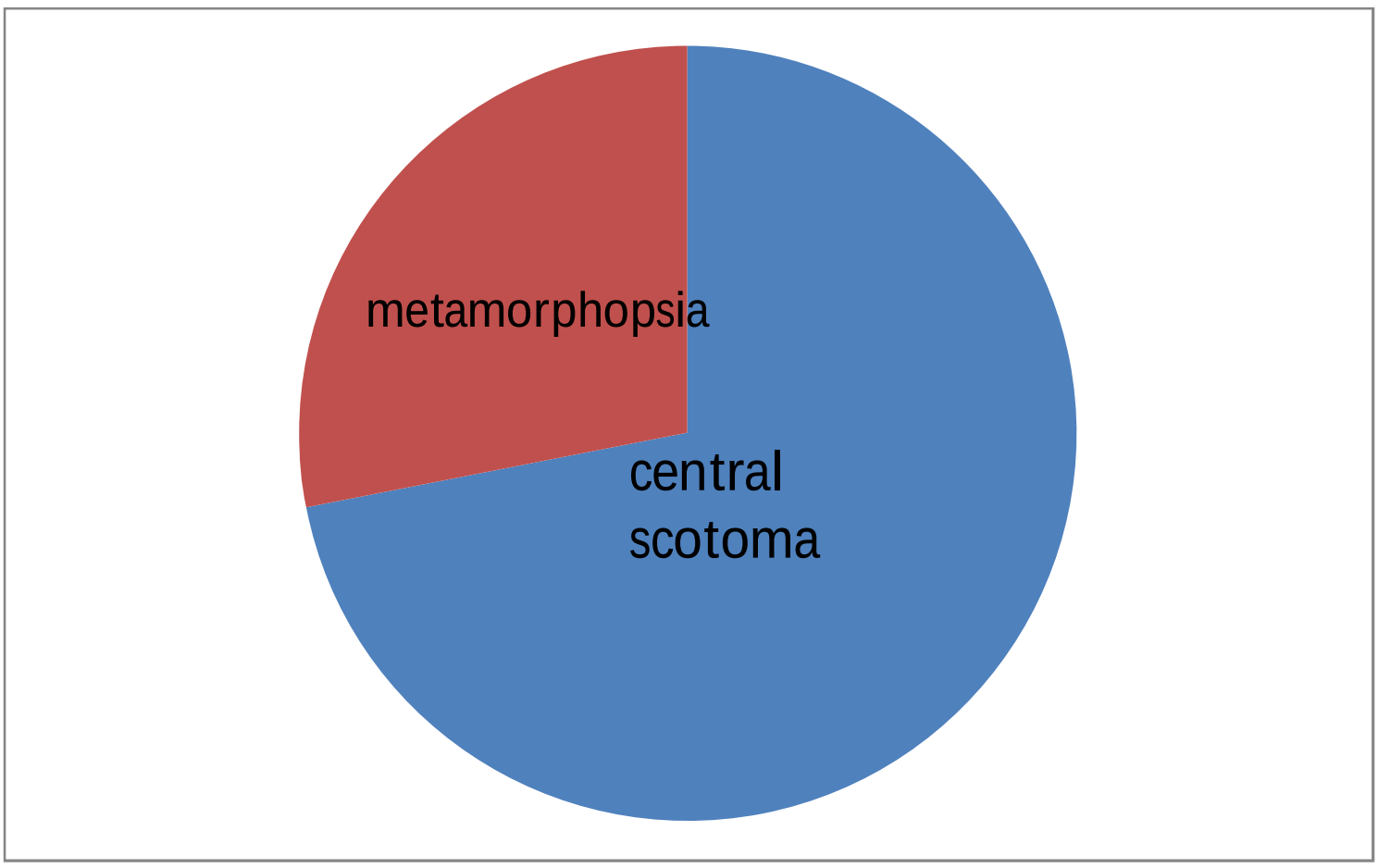
In our study though the incidence of Central serous retinopathy was more in the left eye (52%), there was no significant difference between the eyes involved and there is no predilection for the eye involvement.

4. PRESENTING COMPLAINTS

Table - 4

All patients presented with defective vision

Associated findings	Percentage
Central scotoma	30 (60%)
Metamorphopsia	20 (40%)
Micropsia	0

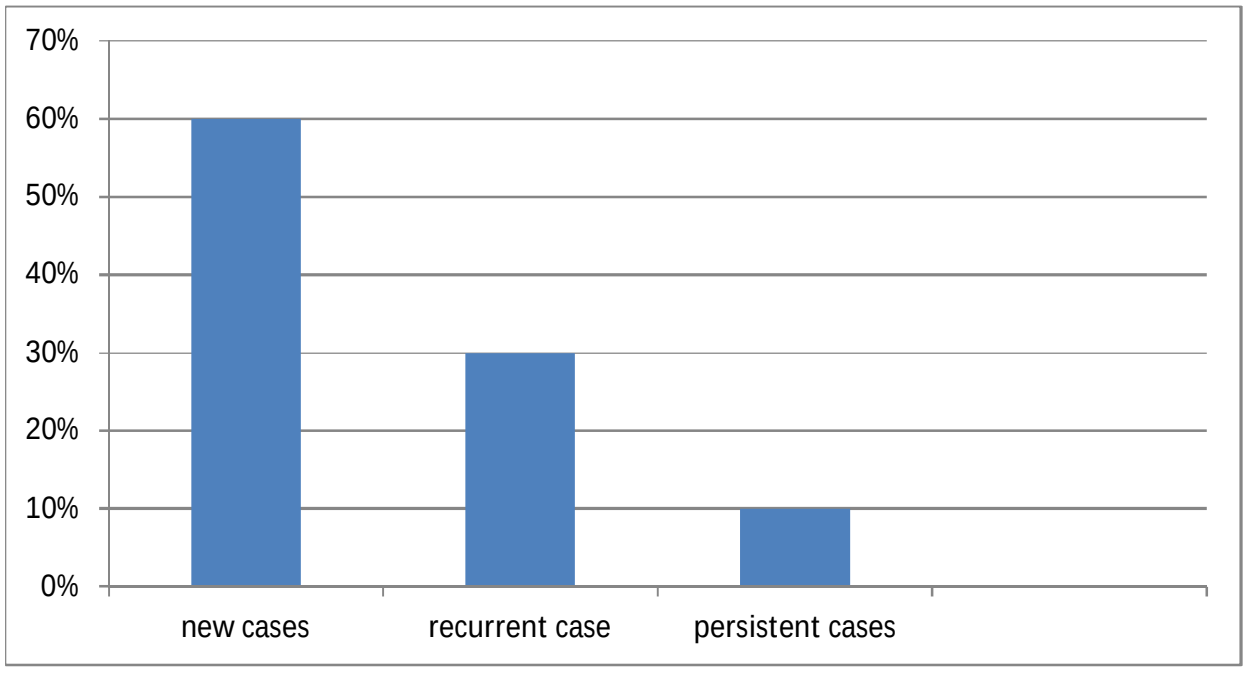


In 60% of patients central scotoma was the predominant presenting complaint.

5. EPIDEMIC INDICES

Table - 5

EPIDEMIC INDICES	NO	PERCENTAGE
Newly diagnosed	30	60%
Recurrent Cases	15	30%
Persistent Cases	5	10%

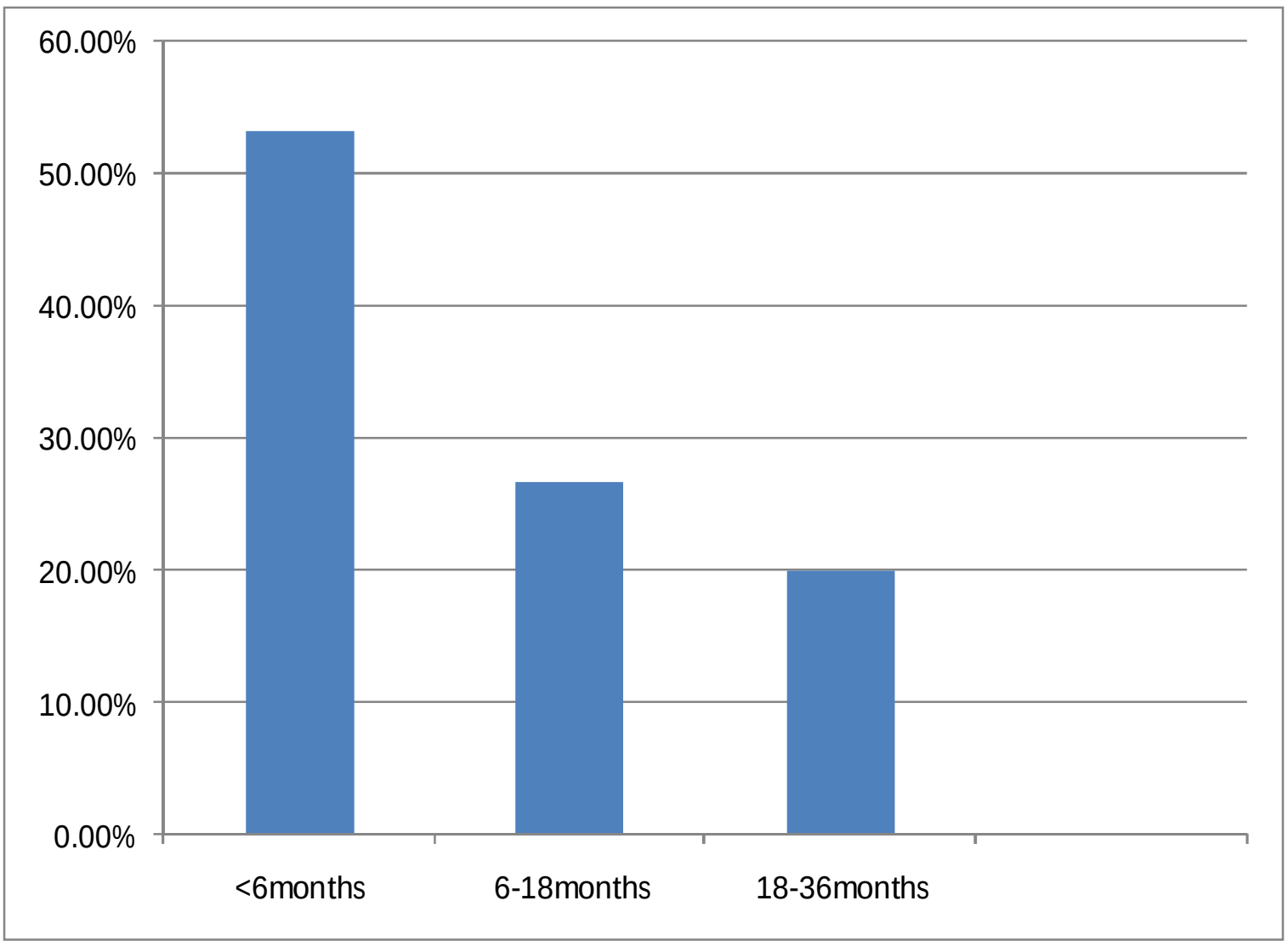


In our study the incidence of first onset of CSCR was 60% and recurrence of the disease in the same or other eye was 30%.In 10% the disease was present for more than 4 months. Gass et al., observed recurrence in 20-30% of the patients.[11]

6. INTERVAL

Table - 6

INTERVAL	NO	PERCENTAGE
<6months	8	53.33%
6months-18months	4	26.67%
18months-36months	3	20%

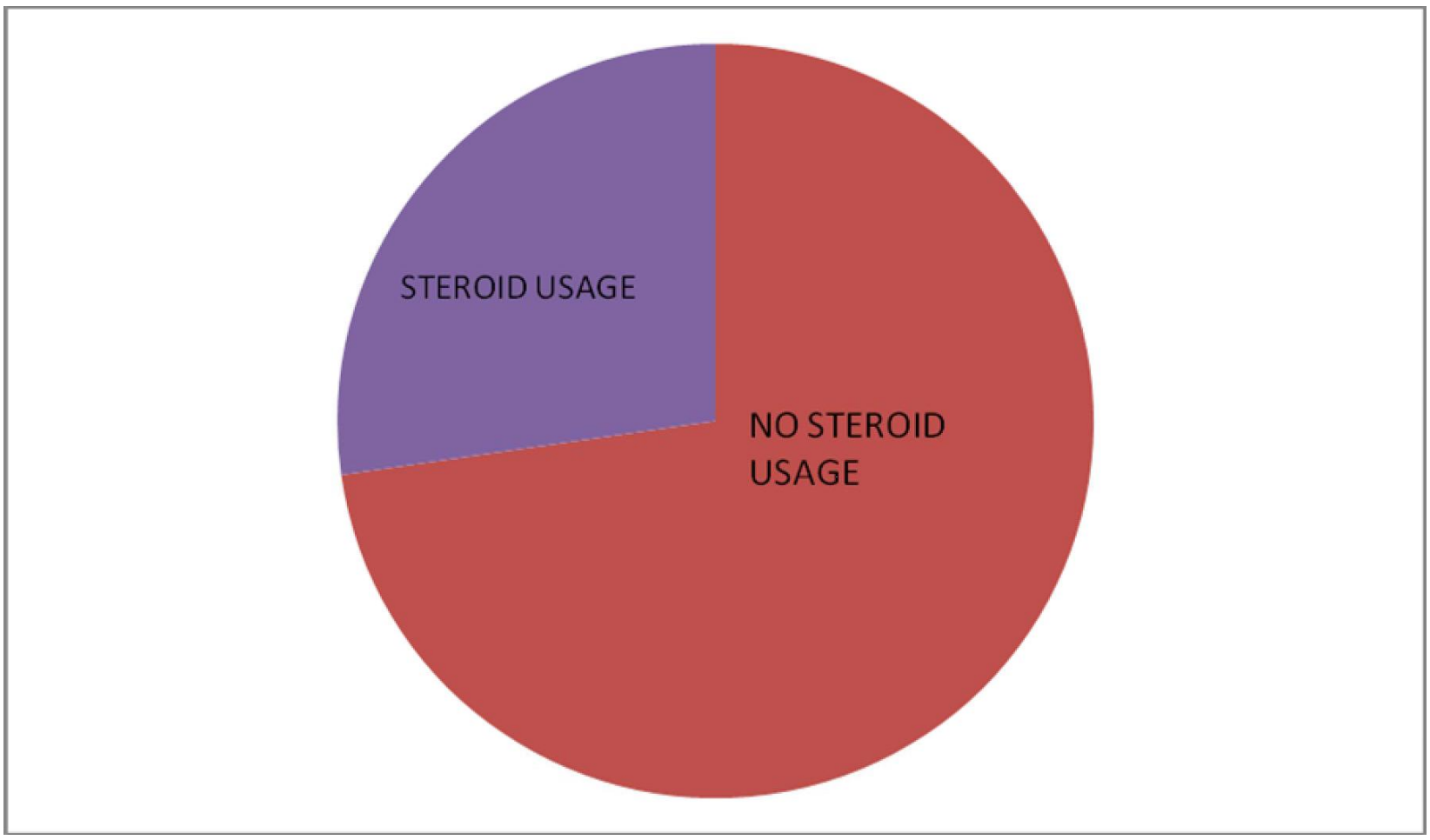


Recurrence rate of CSCR within a period of 6 months was seen in 53.33%.

7. ASSOCIATION WITH STEROIDS

Table - 7

STEROID USAGE	NO	PERCENTAGE
PRESENT	5	10%
ABSENT	45	90%

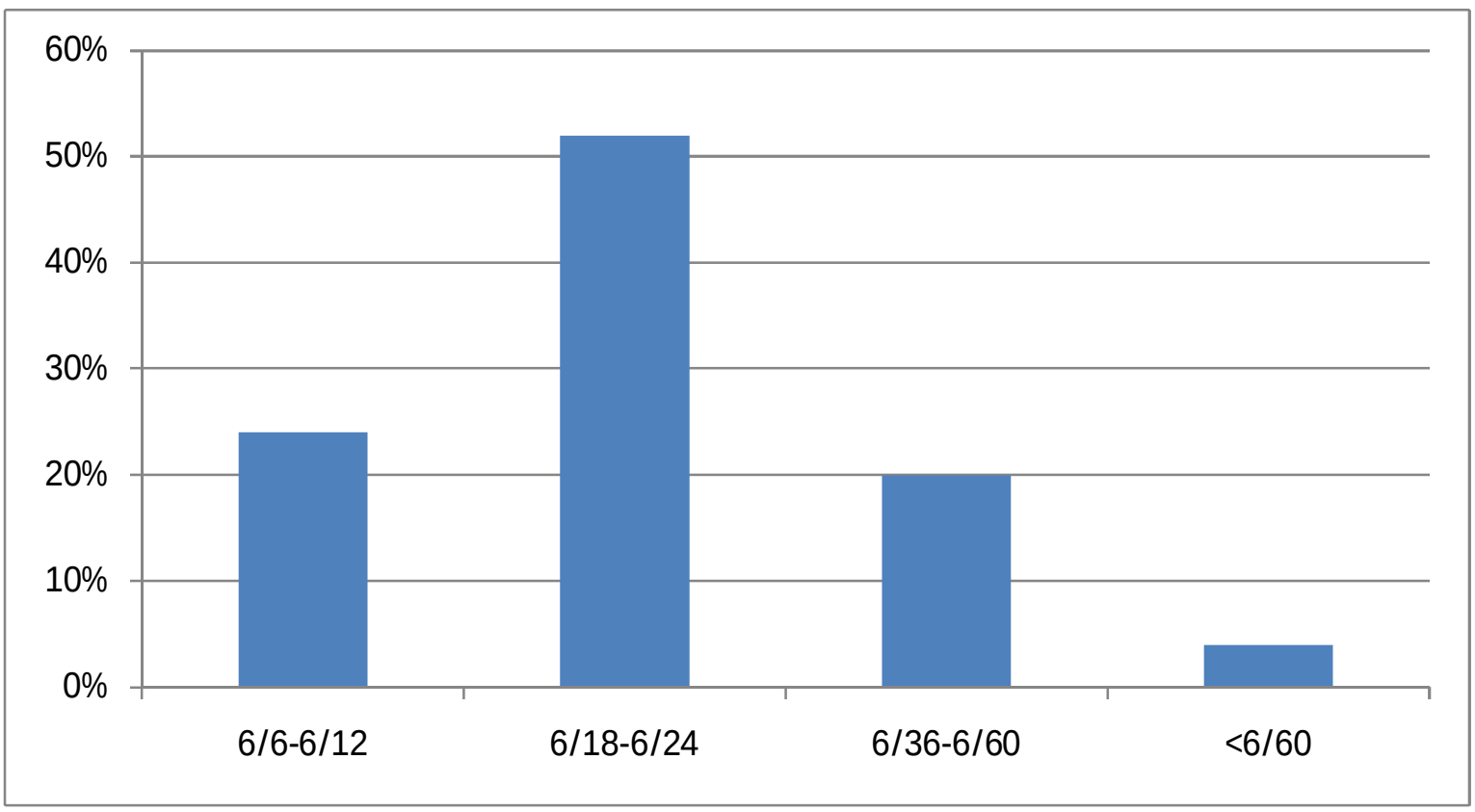


Steroid usage in our study was noted in only 10% of patients. Heimovici R,GragsudassEs, Dukes JS,Sjaarda RN,Eliott observed the association of steroids in CSCR patients.[23]

8. VISUAL ACUITY

Table - 8

VISION	NO	PERCENTAGE
6/6-6/12	12	24%
6/12-6/24	26	52%
6/24-6/60	10	20%
6/60	2	4%

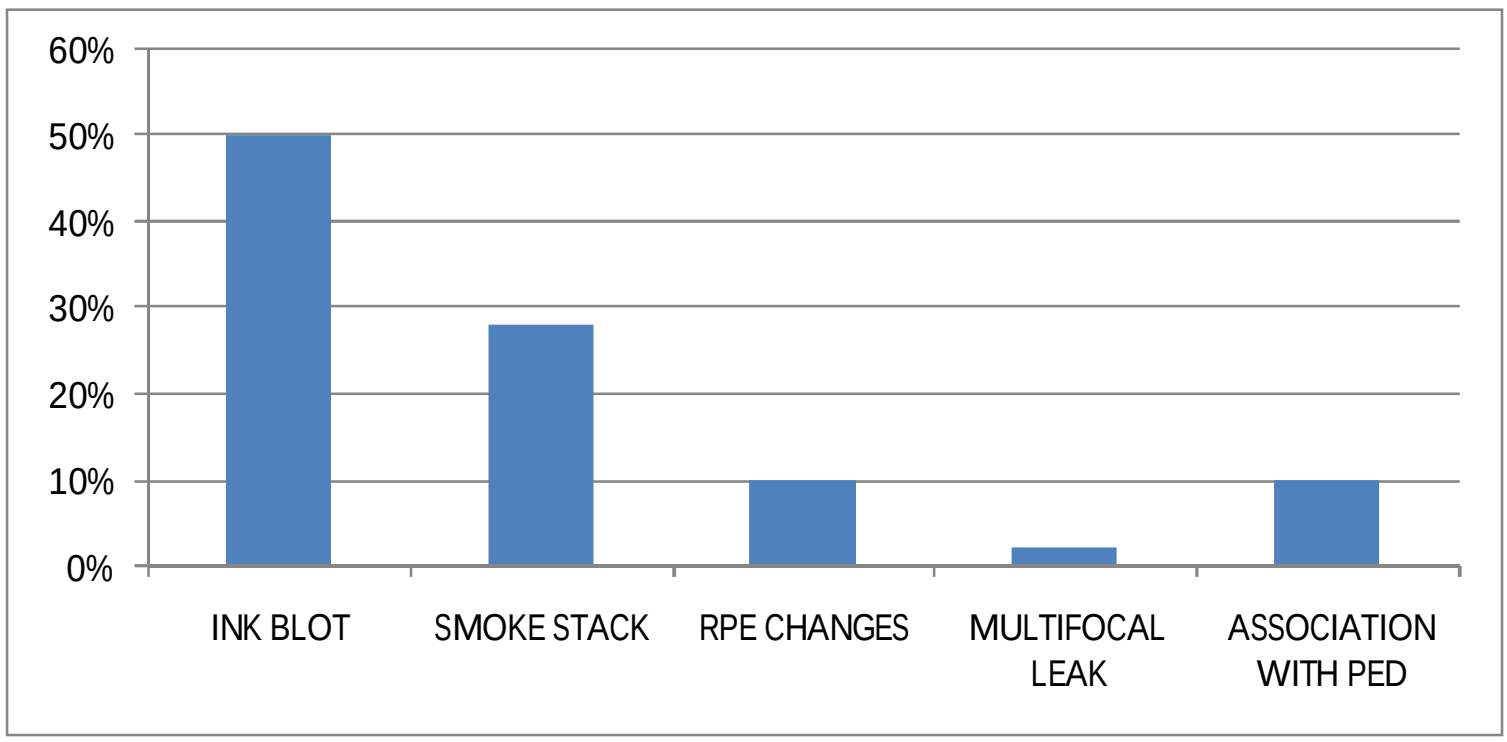


Visual acuity of <6/12 was seen in 24% of patients at presentation

9. FUNDUS FLUORESCEIN ANGIOGRAPHY PATTERNS

Table - 9

FFA PATTERN	NO	PERCENTAGE
INK BLOT APPEARANCE	25	50%
SMOKE STACK APPEARANCE	14	28%
RETINAL PIGMENT EPITHELIAL CHANGES WITH NO LEAK	5	10%
ASSOCIATION WITH PED	5	10%
MULTIFOCAL CSR	1	2%

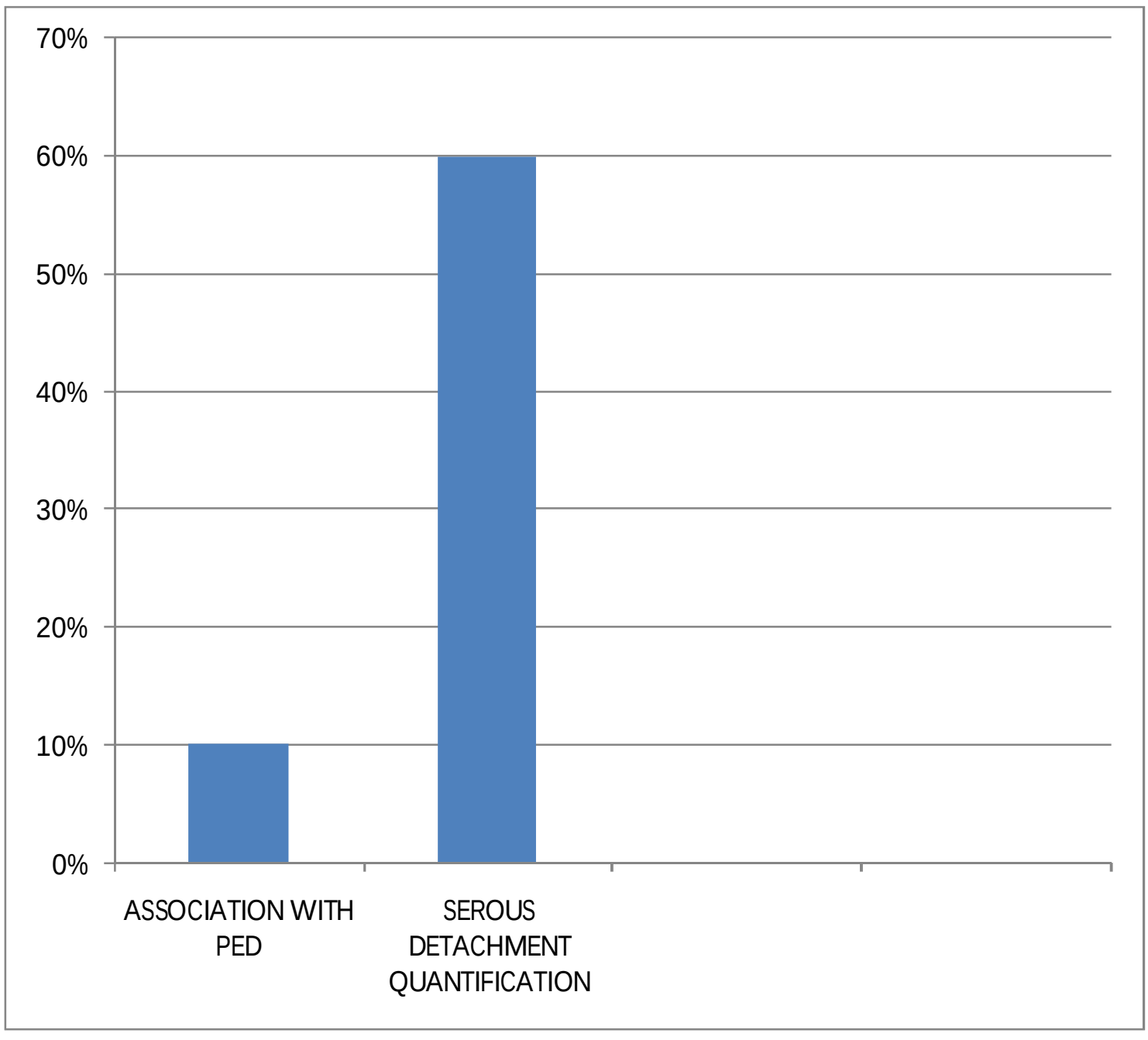


Ink blot pattern is the predominant pattern in 50% of patients.

10. OPTICAL COHERENCE TOMOGRAPHY

Table - 10

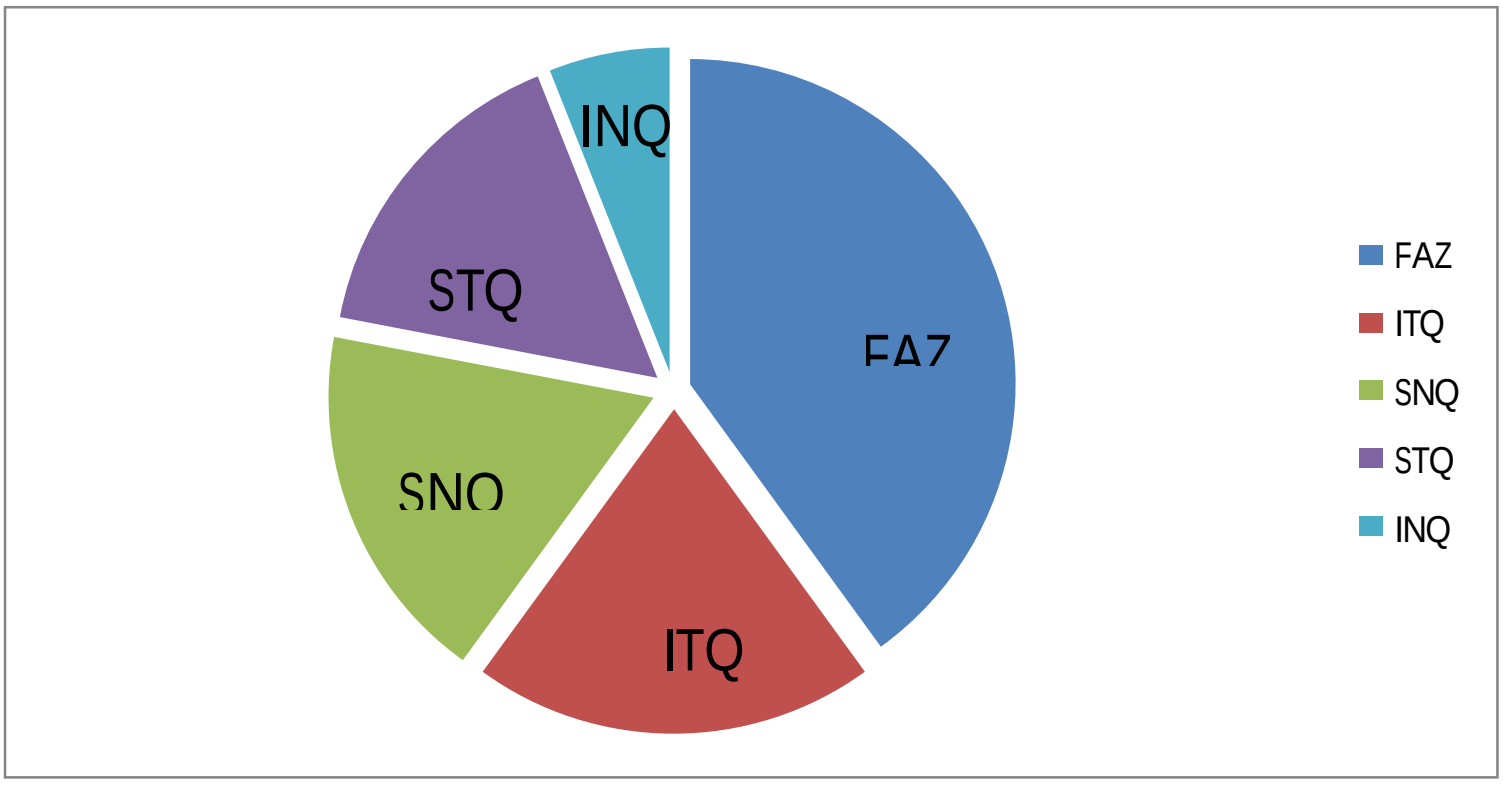
OCT PATTERN	NO	PERCENTAGE
ASSOCIATION WITH PED	6	12%
SEROUS DETACHMENT QUANTIFICATION	30	60%



11. LOCATION OF LEAK

Table - 11

SITE OF LEAK	NO	PERCENTAGE
FAZ	20	40%
STQ	8	16%
SNQ	9	18%
ITQ	10	20%
INQ	3	6%



Leakage within 500µm of fovea was seen in 40% of patients. Bennet, G., observed that the overall incidence of leakage points was greatest in the upper nasal quadrant, followed by lower nasal quadrant, the upper temporal quadrant and the lower temporal quadrant,in decreasing order of frequency.[13]

SUBGROUP ANALYSIS

TREATMENT

30 patient with first onset of Central Serous Chorioretinopathy for whom the FFA and OCT showed evidence of leakage 375 µm away from fovea were randomly divided into two groups .

Subgroup 1

15 randomly selected patients were subjected to laser photocoagulation with Semiconductor green dye laser of 532nm with laser parameters

- 100µm-200µm spot size,
- 100mw-150mw power,
- 0.1-0.2 seconds duration.

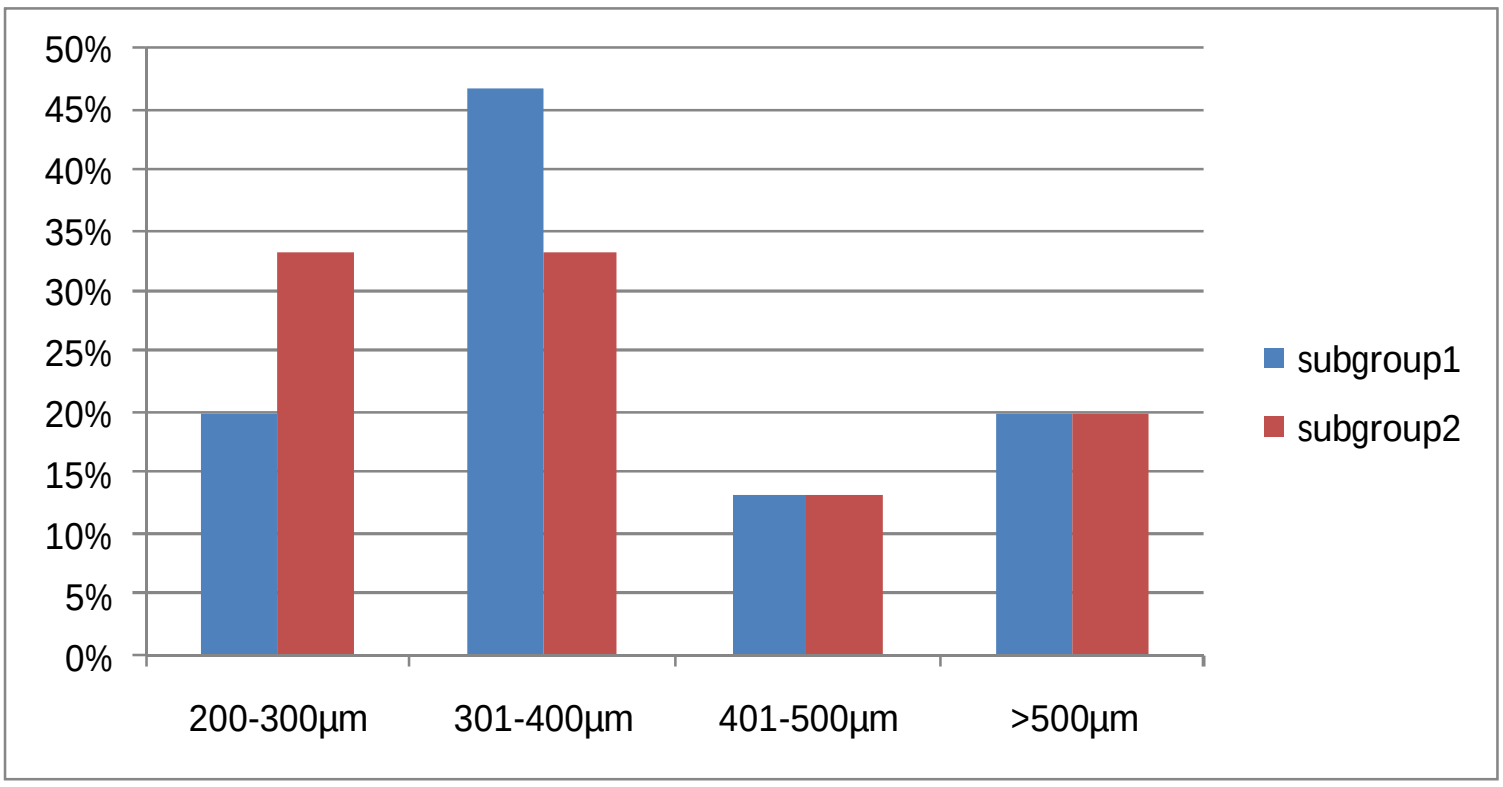
Subgroup 2

The remaining 15 patients were given placebo treatment and observed. Visual acuity using snellens chart and Central macular thickness of serous detachment using OCT of both group was observed before and after laser treatment and patients were followed up at 4^{th} and 12^{th} week.

12. MACULAR THICKNESS PRE-TREATMENT (Study and control group)

Table - 12

MACULAR THICKNESS	PRE-TREATMENT SUBGROUP 1	PRE-TREATMENT SUBGROUP 2
200-300µm	3(20%)	5(33.33%)
301-400 µm	7(46.67%)	5(33.33%)
401-500 µm	2(13.33%)	2(13.33%)
>500 µm	3(20%)	3(20%)

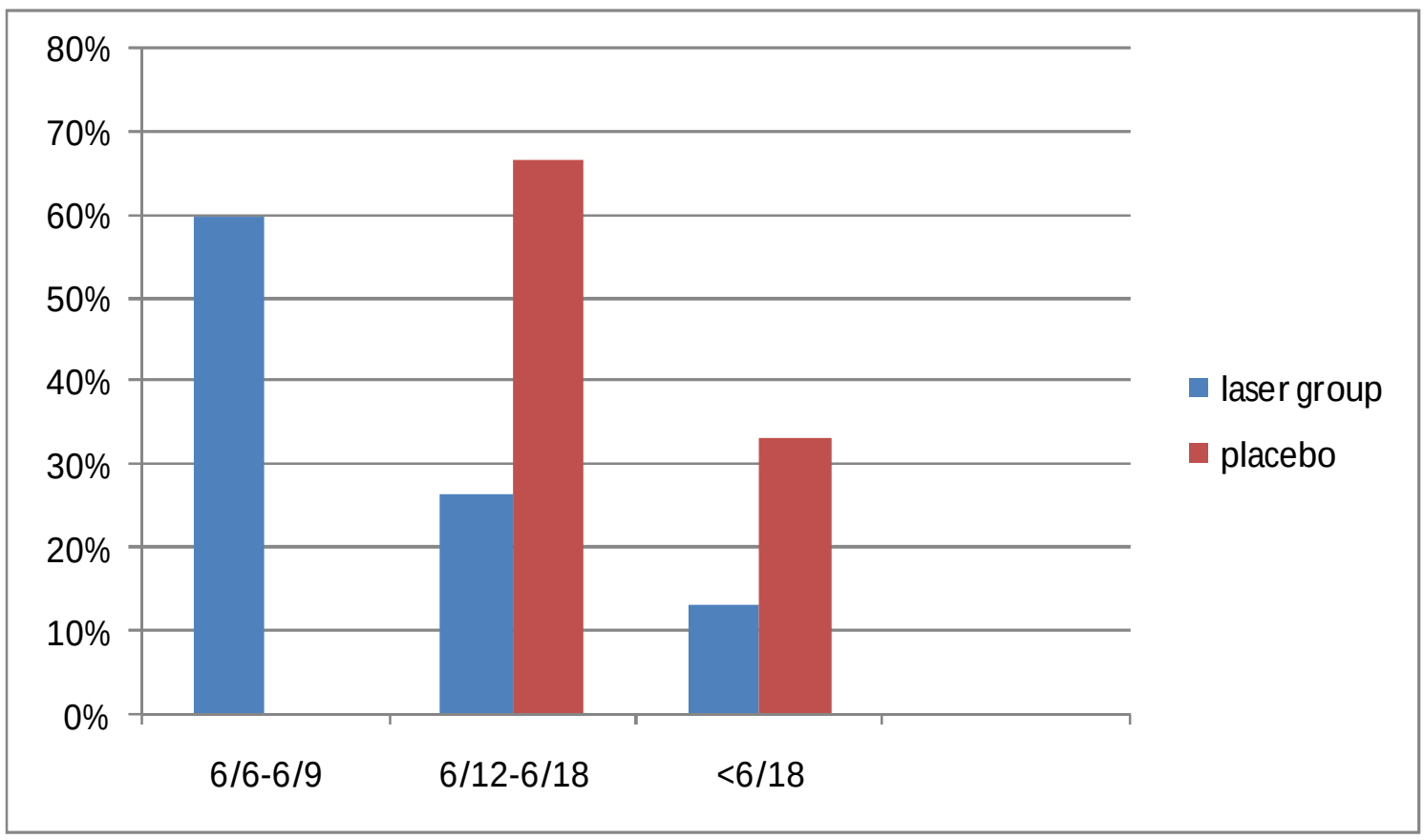


The average pretreatment macular thickness in the subgroup 1 was 403.60 µm and in subgroup 2 was 421.87µm.The predominant range of thickness in the two groups were 301-400µm.

13. VISUAL ACUITY POST TREATMENT AT 4TH WEEK

Table - 13

VISUAL ACUITY	LASER GROUP	PLACEBO
6/6-6/9	5(33.33%)	0
6/12-6/18	9(60%)	10(66.67)
<6/18	1(6.667%)	5(33.33%)

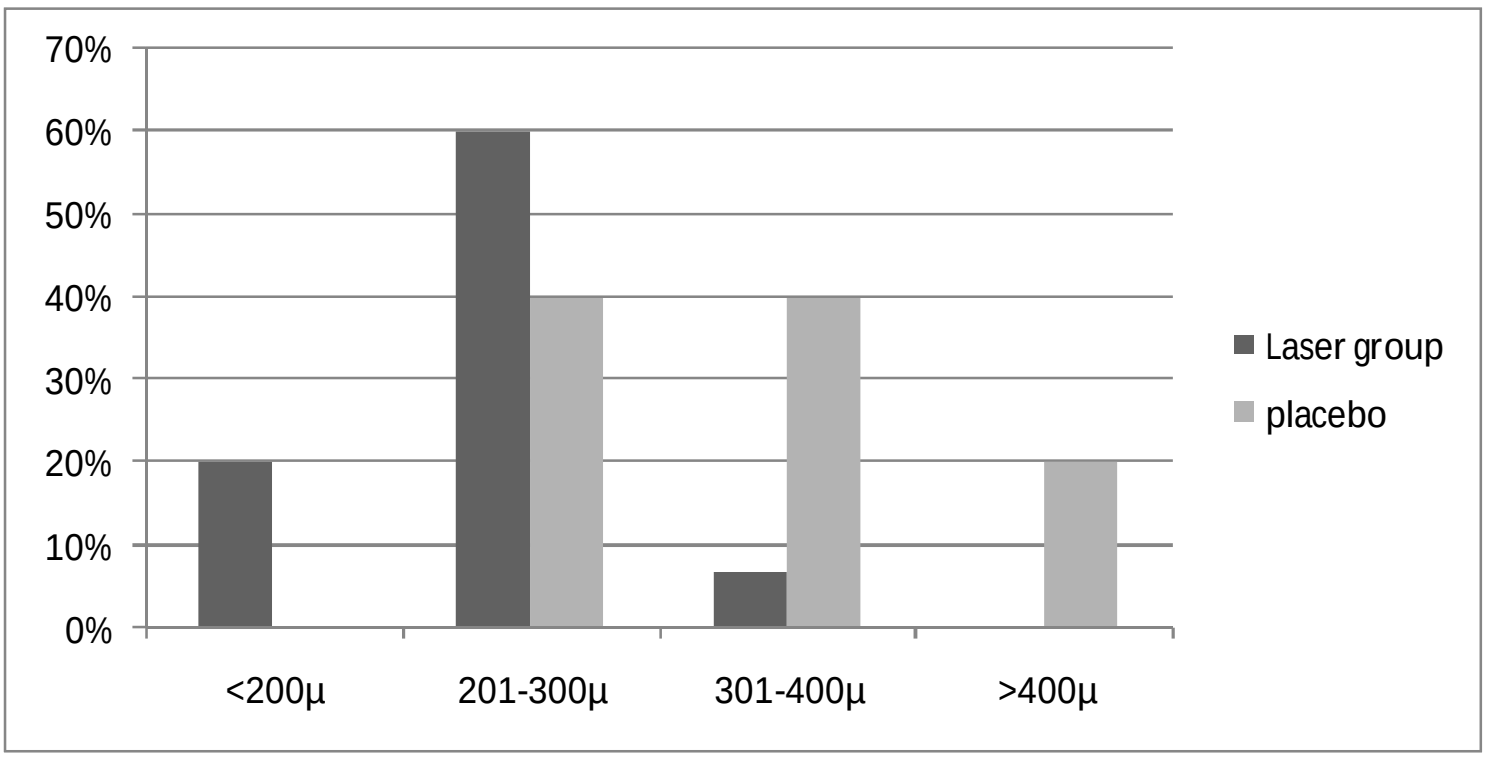


Best corrected Visual acuity of 6/6-6/9 was seen in 33.33% of laser group 0%in the observation group.

14. POST TREATMENT MACULAR THICKNESS AT 4TH WEEK

Table - 14

MACULAR THICKNESS in µm	LASER GROUP	PLACEBO
<200	3(20%)	0
201-300	10(66.67%)	6(40%)
301-400	2(13.33%)	6(40%)
>400	0	3(20%)

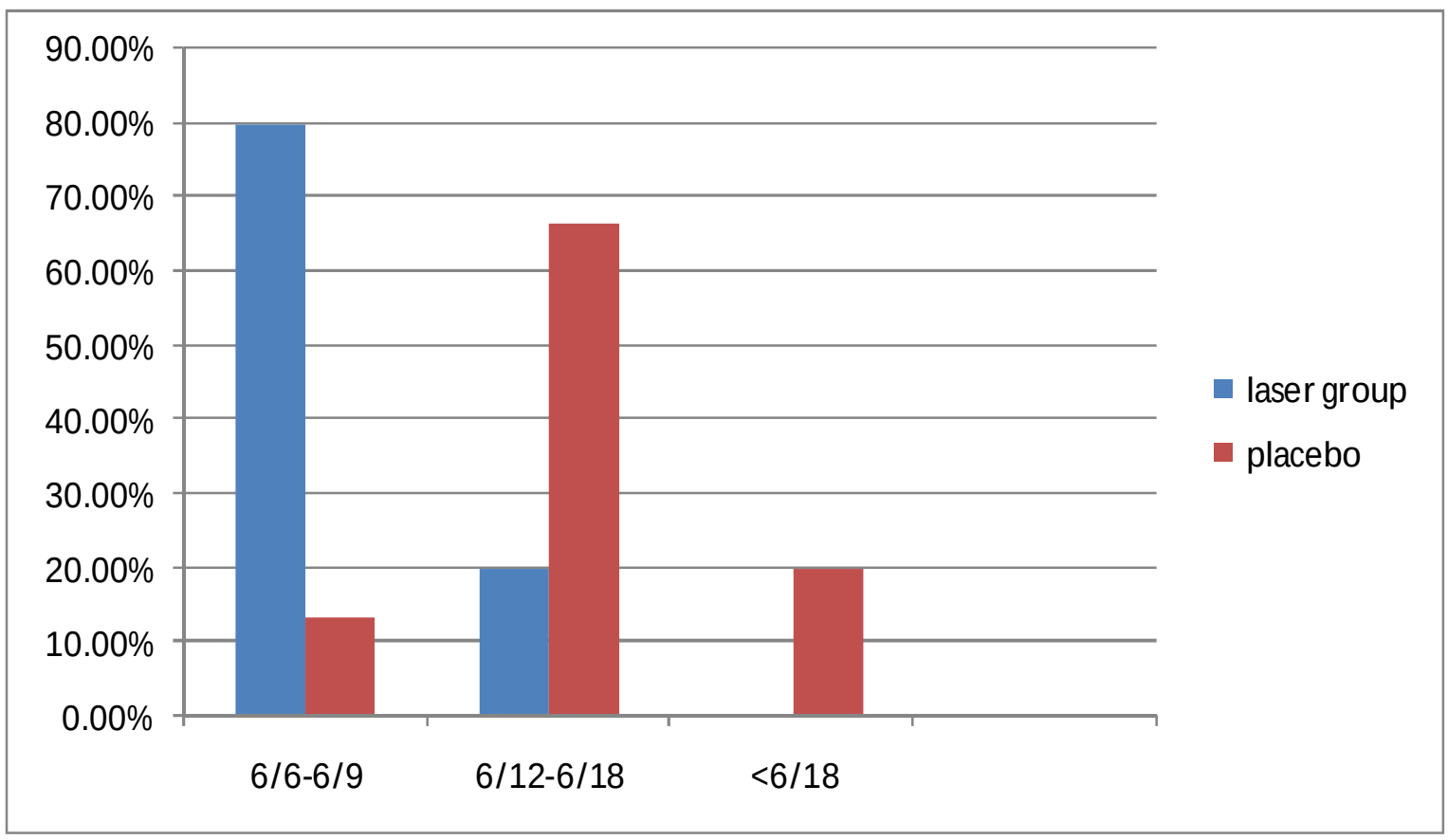


Average reduction in the central macular thickness in subgroup 1 at the end of 4th week was 157µm, whereas in subgroup 2, it was 56.87µm.

15. VISUAL ACUITY AT 12TH WEEK

Table - 15

VISUAL ACUITY	LASER GROUP	PLACEBO
6/6-6/9	12(80%)	2(13.33%)
6/12-6/18	3(20%)	10(66.67%)
<6/18	0%	3(20%)

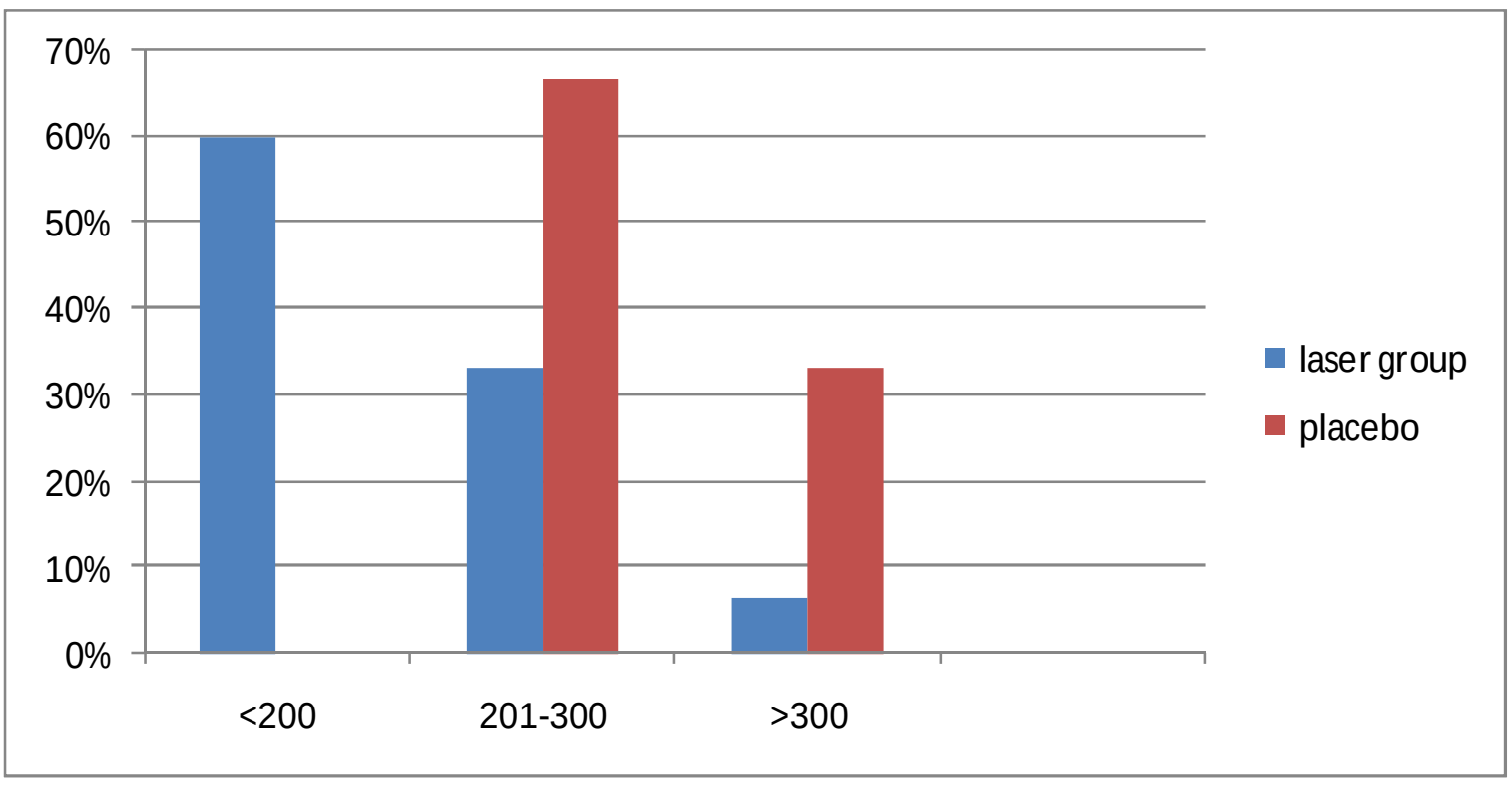


Best corrected visual acuity of 6/6-6/9 was noted in 80% in the subgroup 1, whereas only 13.33% had visual acuity of 6/6-6/9 in subgroup 2.

16. MACULAR THICKNESS POST TREATMENT AT 12TH WEEK

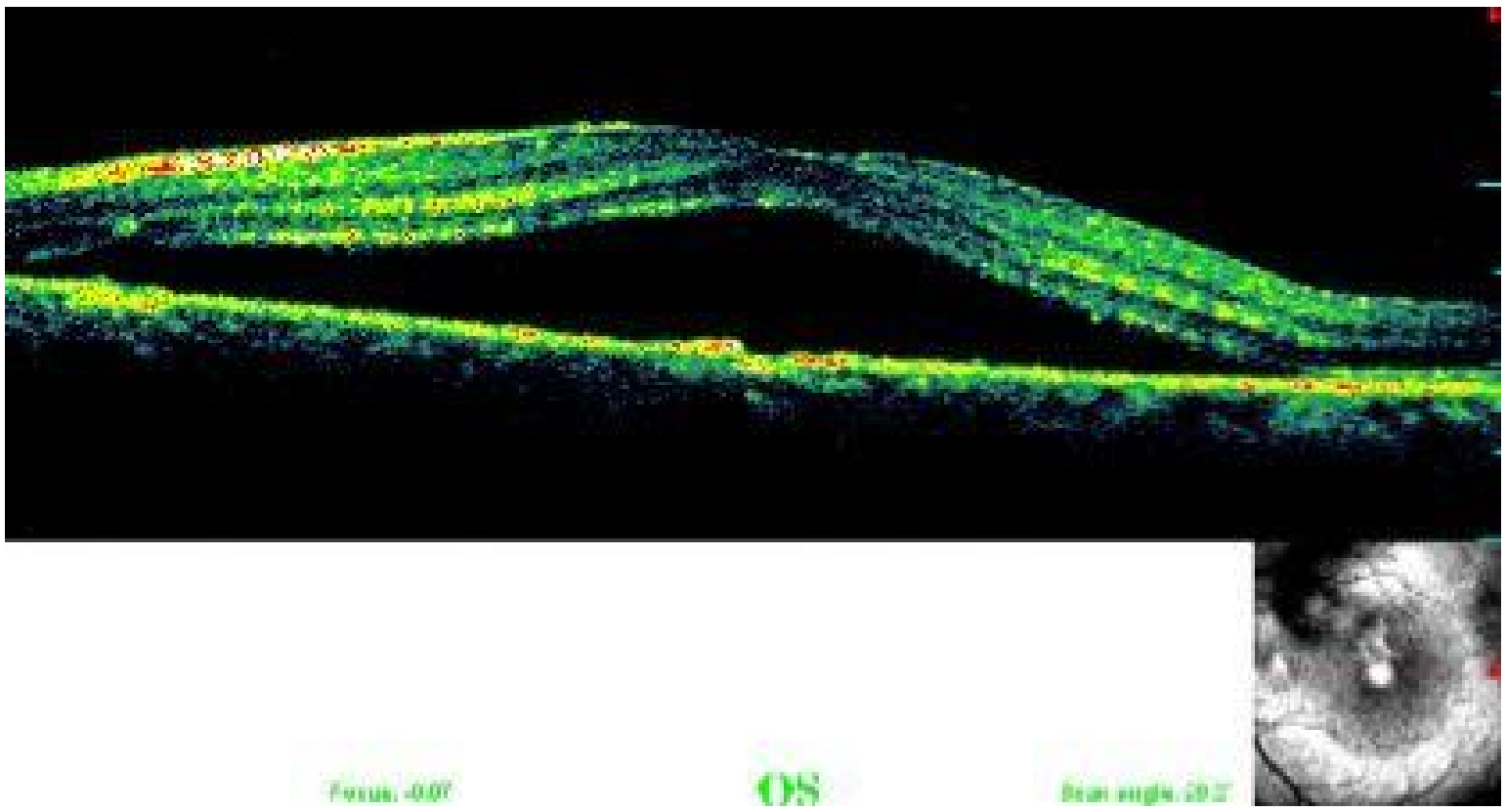
Table - 16

MACULAR THICKNESS	LASER GROUP	PLACEBO
<200	9(60%)	
201-300	5(33.33%)	10(66.66%)
>300	1(6.66%)	5(33.33%)

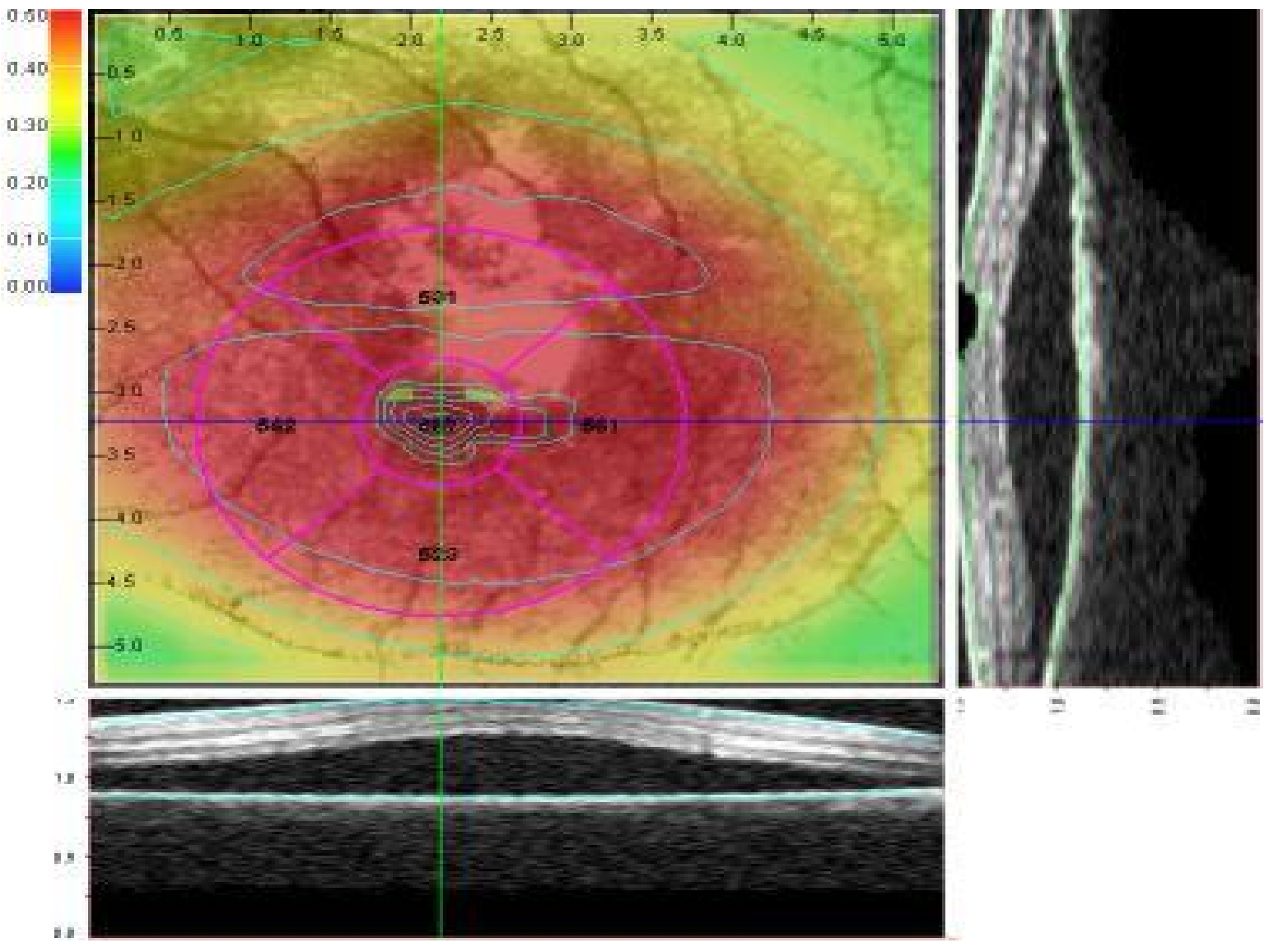


Average reduction in the central macular thickness in subgroup 1 at the end of 12th week was 186µm,whereas in subgroup 2, it was 98.87µm.

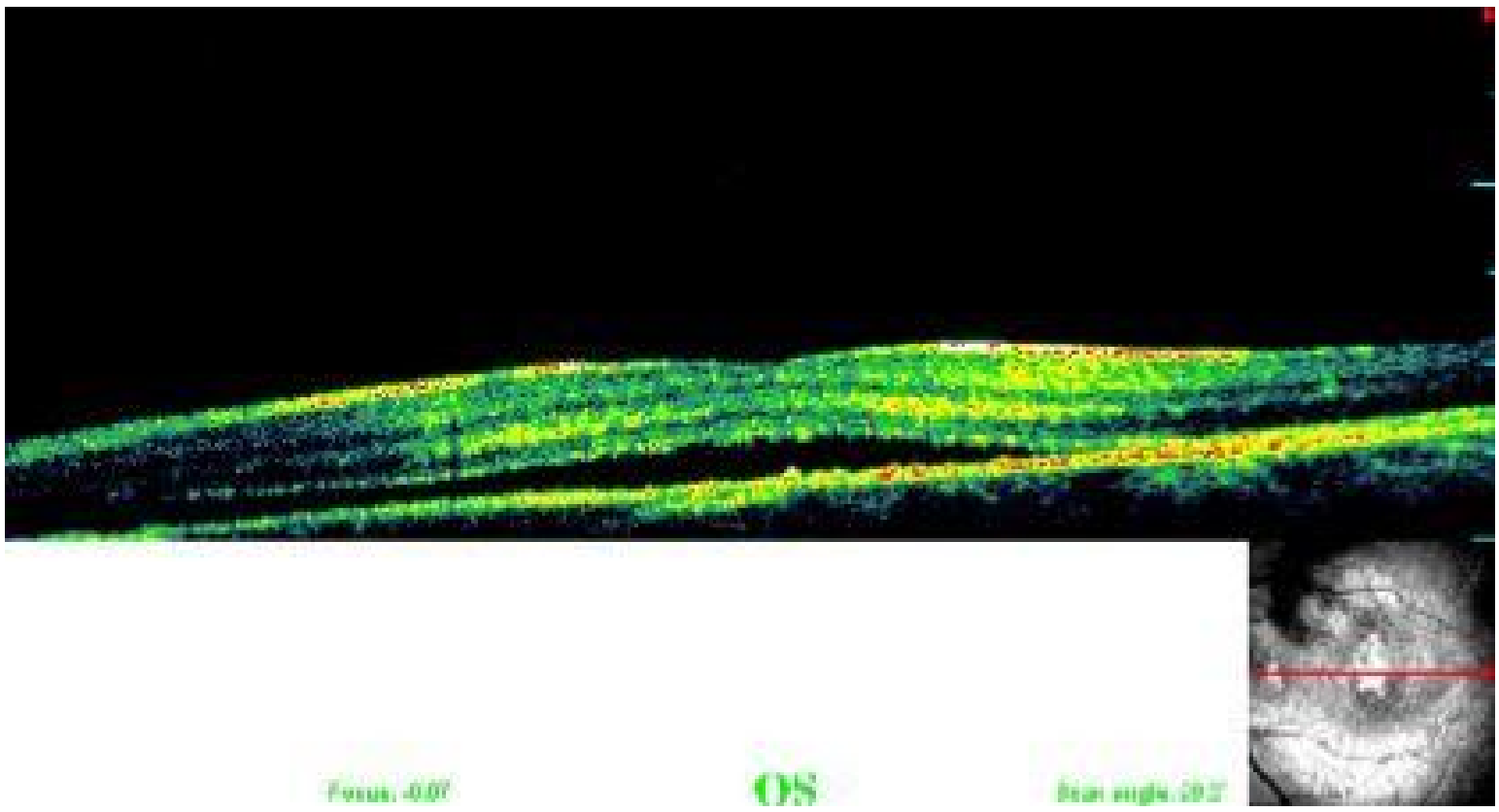
PATIENT VELMURUGAN OS-CSCR
PRE-TREATMENT THICKNESS



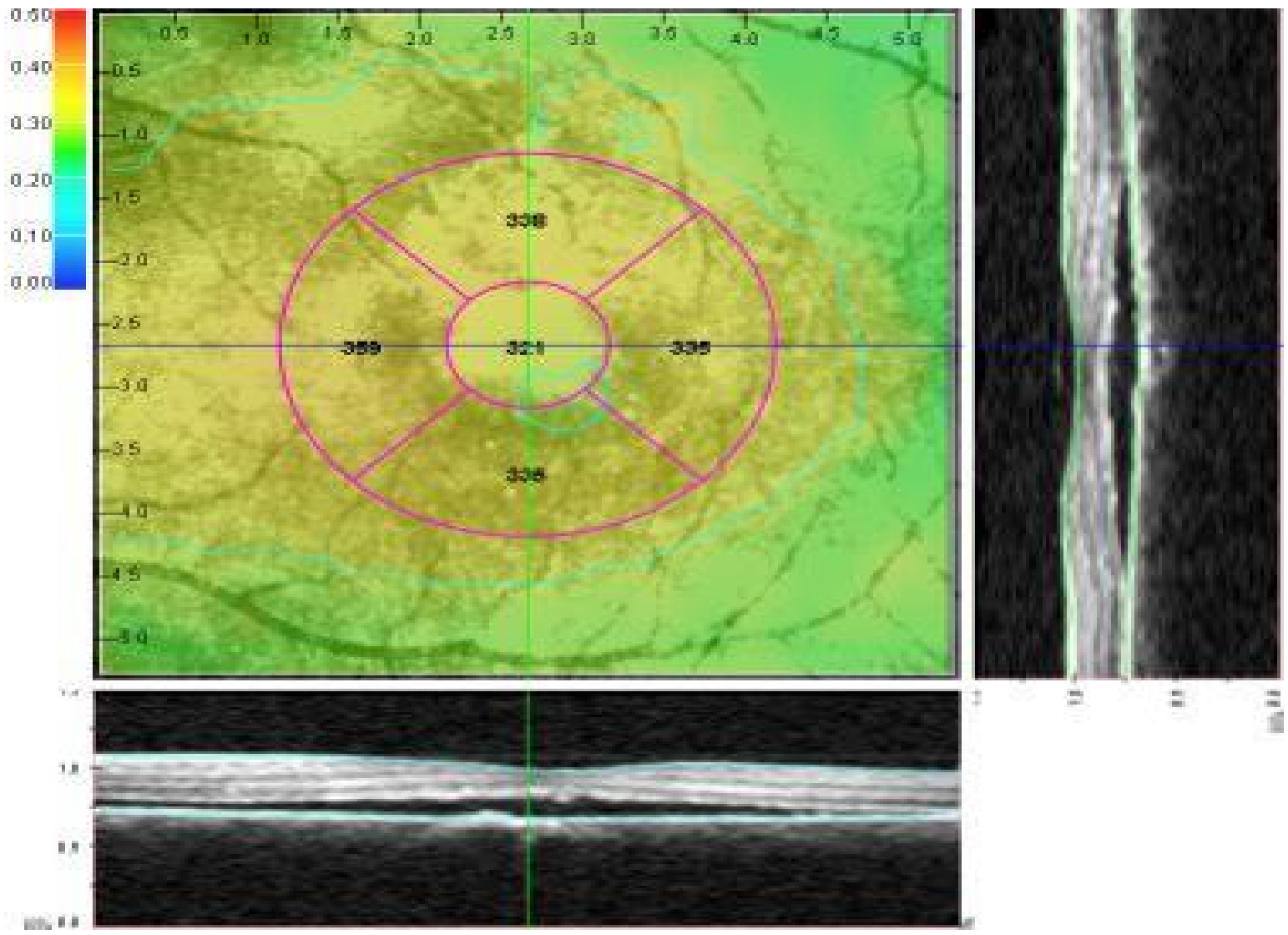
LE - TOPOGRAPHY



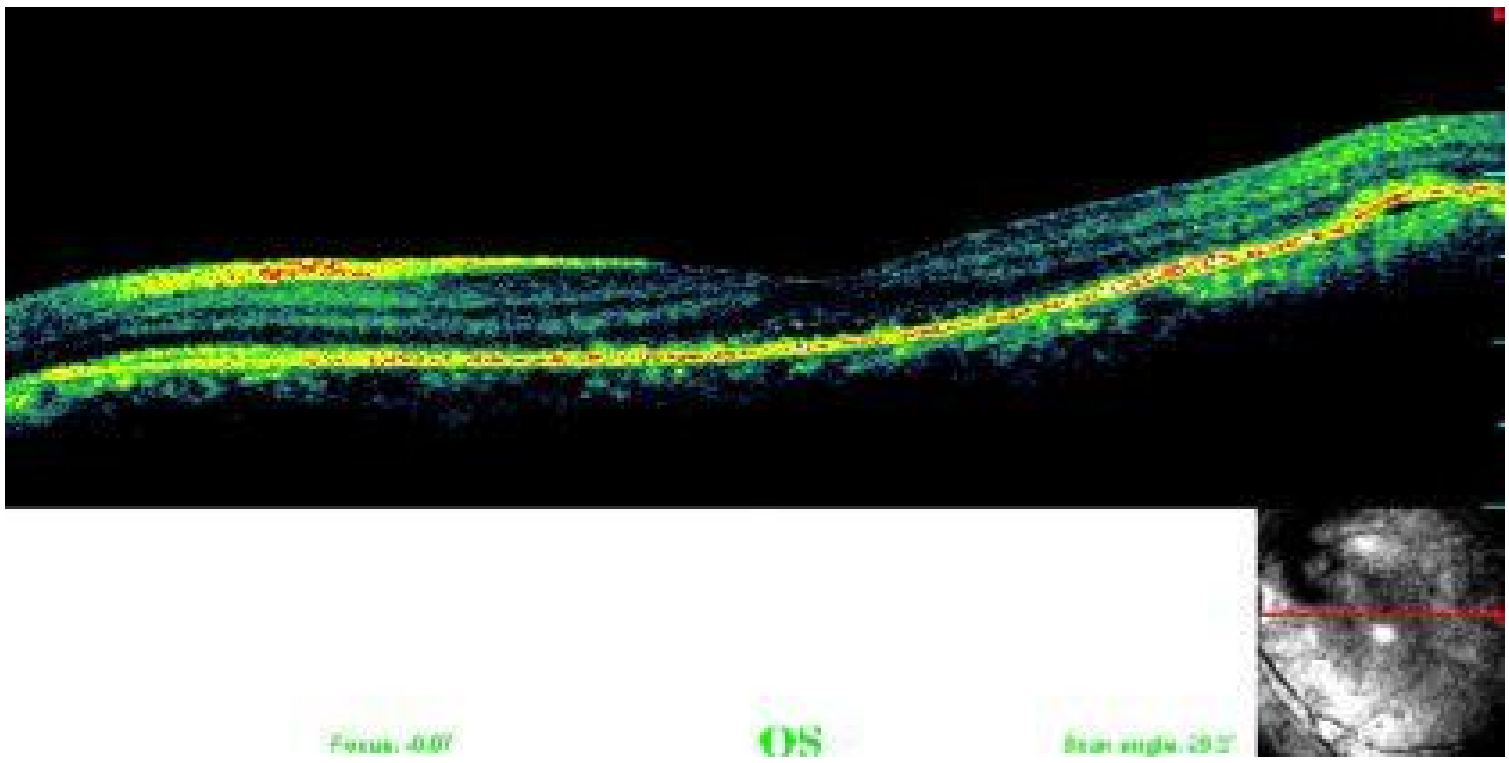
VELMURUGAN-OS-CSCR
POST-TREATMENT THICKNESS 4TH WEEK



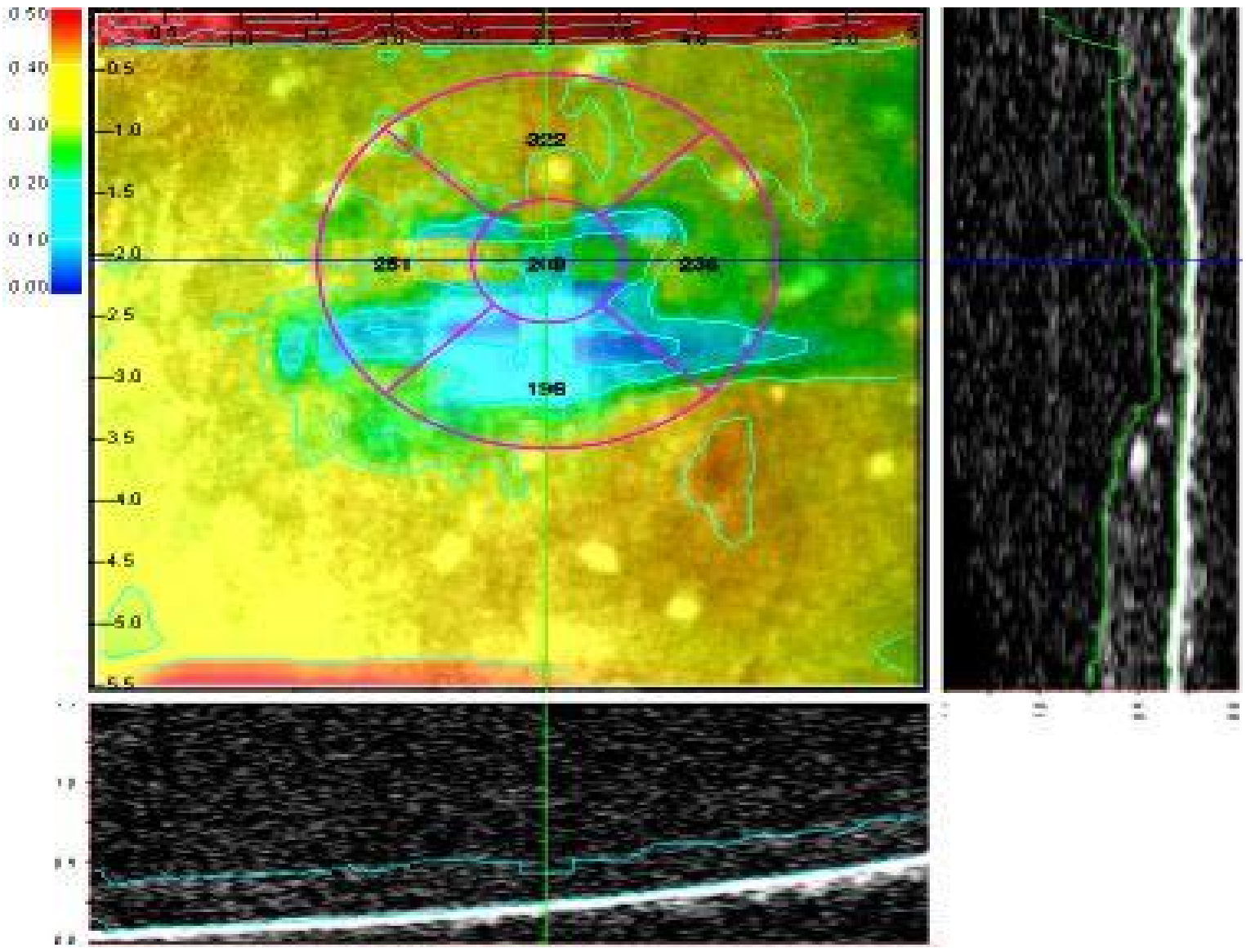
LE - TOPOGRAPHY



VELMURUGAN-OS-CSCR
POST-TREATMENT 12TH WEEK



LE - TOPOGRAPHY



DISCUSSION AND RESULTS

1. In our study of 50 patients the age group affected was between 20 and 50 years. Previous studies showed involvement between 20 and 50years of age (Gass,et.al).[11]

2. Male:female ratio was 9:1.

David A.Quillenet al., observed that women tend to be older at the time of onset when compared to men, since in our study only one female has been reported and the difference in age groups cannot be found out so this emphasis could not be proved.

3. In our study though the incidence of Central serous retinopathy was more in the left eye (52%), there was no significant difference between the eyes involved and there was no predilection for the eye involvement. 4% of the patients had bilateral involvement.

4. All patients presented to us with defective central vision, about 60% of patients gave characteristic central scotoma, remaining 40% patient had metamorphopsia.

5. The recurrence rate of CSCR in our study was seen in 30% of the patients and the interval between the previous and the present episode ranged from 4 months to 3 years.

   About 53.33% of patients had recurrence within the period of 6 months. About 10% of patients had persistent disease of duration >4months. The incidence of recurrence was more in patient with mental stress though it could’nt be quantified.

   Gass et al observed recurrences in 20-30% of patients.[11]

6. History of steroid usage as systemic, topical and ointment form was seen in 10% patients. The remaining 90% gave no history of steroid usage but had history of increased mental stress.

7. The best corrected visual acuity at the time of presentation ranged from 6/6-6/60.

   In 52% of patients the corrected visual acuity was between 6/12-6/24.

8. Various Fundus fluorescein angiographic patterns were studied, of which the Ink blot pattern was seen in 50%, smoke stack pattern in 28%, retinal pigment epithelial changes seen in 10%,

pigment epithelial detachment association was seen in 10%, 2% (one patient) had multifocal leakage and there was a history of steroid usage in him.

9. Location of leakage in fluorescein angiography showed that 40% was within the foveal avascular zone or within 375µm from the centre of fovea, 20% in the inferotemporal quadrant of macula, In 18% the leakage was in the superonasal quadrant, In 16% the leakage in the superotemporal quadrant and in 6%, the inferonasal quadrant.

Bennet, G., observed that the overall incidence of leakage points was greatest in the upper nasal quadrant, followed by lower nasal quadrant, the upper temporal quadrant and the lower temporal quadrant in decreasing order of frequency.[13]

10. Optical coherence tomography was helpful in quantification of serous detachment. Central Macular thickness was 200-300µm in 26.66% of the patients, in about 46.66% thickness ranged 301-400µm, in 6.66% the thickness was 401-500µm, in about 20% the macular thickness was about >500µm. It was further observed

that pigment epithelial detachment was present in only 5% of the patients.

Although association of PED facilitated the pathogenesis of central serous chorioretinopathy. It was not observed in majority of patients studied.

11. Subgroup 1, 15 patients with leak 375µm away from the fovea were treated with laser photocoagulation. It was observed that 60% regained 6/6-6/9 vision within a period of 4 weeks whereas no (0%) patient in subgroup 2 had 6/6-6/9 vision. This study is supported by Early laser photocoagulation in patients with CSCR, by V.Strum, R.Schwartz & G.Richard Ophthalmology university of Hamburg,Germany.[30]

12. Visual acuity observed at 12 weeks showed that 73.33% regained 6/6-6/9 vision whereas only 13.33% patients in control group regained 6/6-6/9 vision. This observation is supported by Early Laser photocoagulation in patients with CSCR, Department of Ophthalmology, Hospital of Zurich, Switz.

13. Average reduction in the central macular thickness in subgroup 1 at the end of 4th week was 157µm, whereas in subgroup 2, it was 56.87µm.

14. Average reduction in central macular thickness in the subgroup 1(study group) at the end of 12th week was 186µm, whereas in subgroup 2, it was 98.87µm.

15. About 60% of patients in the sub group 1 had complete resolution of serous detachment at the end of 12 weeks, whereas the remaining 40% had residual detachment inspite of response to treatment due high pretreatment macular thickness.

16. Thickness in the range of 201-300µ was noted in 66.67% of the Subgroup 2.No patient had complete resolution at the end of 12 weeks as compared to laser treatment group.

17. The statistical analysis for the macular thickness at 4 weeks showed significant probability **chi-square test of 0.029% and o.oo2% at 12 weeks**

18. The statistical analysis for the visual acuity at 4 weeks showed significant probability **chi-square test of 0.041% and 0.017%** at 12 weeks.

CONCLUSION

CENTRAL SEROUS CHORIORETINOPATHY predominantly affects healthy male (90%).

Ink blot pattern is the most common fluorescein pattern found in the study (50%).

OCT helps in quantification of macular thickness and look for association of pigment epithelial detachment and choroidal neovascularisation.

Early Laser photocoagulation is effective in rapid restoration of vision and reduction in macular thickness within a period of 12 weeks, and there is no complication reported in our study due to laser treatment.

Photocoagulation play major role in rapid restoration of vision 6/6-6/9 within period of 12 weeks in about 73.33%, whereas only 13.33% of the patient in placebo group had 6/6-6/9 vision.

Early laser photocoagulation results in rapid visual restoration within a period which ranged from 2-12weeks.

The recurrence rate of CSCR in our study was 30% and the interval between previous and present episodes ranged from 4 months to 3 years by prospective and retrospective analysis. About 10% of patients had persistent disease of duration more than 4 months.

Majority of patients gave history of increased mental stress.

History of steroid usage either systemic, topical or inhalers and ointment was noted in minority of patients(10%).

LIMITATIONS

Post laser recurrences of CSCR could not be documented with follow up period of 4^{th} weeks and 12^{th} weeks. Furthermore late post laser complications such as CNVM could be identified only by extended follow up.

PART THREE

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PROFORMA

NAME

AGE/SEX **SERIAL NO**

OP NO

ADDRESS

PHONE NO

OCCUPATION

COMPLAINTS

Defective vision

Central scotoma

Metamorphopsia/Micropsia

Headache /others

HISTORY

1. **Past eye illness**

   Similar episode

   Duration of recurrence

2.Personal History

Mental stress **Allergy**

Smoking **DRUG INTAKE**

3.Systemic Illness

EXAMINATION OF EYE

Visual Acuity

Anterior Segment

Tension

Amsler Grid

Fundus examination

Direct Ophthalmoscopy

Slit lamp biomicroscopy with 90D

Fundus Fluorescein Angiography

Pattern- Ink Blot/Smoke Stack/Multifocal Leak

RPE changes

Site Of Leak

FAZ

Elsewhere In Macula

OPTICAL COHERENCE TOMOGRAPHY

Central Macular Thickness

RE/LE

PED - YES/NO

GENERAL EXAMINATION

Anemia

Focal sepsis

ENT

DENTAL

INVESTIGATION

Hb%

TC-

DC-

ESR-

Mx

VDR

TREATMENT

LASER PHOTOCOAGULATION

PLACEBO THERAPY

FOLLOW UP

DATE

VISION

CENTRAL MACULAR THICKNESS

AMSLER GRID CHART

KEY TO MASTER CHART

SEX

M - Male

F - Female

EYE AFFECTED

RE - Right eye

LE - Left eye

D.V - Defective vision

AMSLER'S CHARTING

C.S - Central scotoma

M - Micropsia

MM - Metamorphopsia

PREVIOUS ATTACK

S.E - Same eye

O.E - Other eye

VISUAL ACUITY

NIP - No improvement with pinhole

PH - Pinhole

P - Partial

ATTACK INTERVAL

M	-	Months
S.U.	-	**STEROID USAGE**

FUNDUS FLUORESCEIN ANGIOGRAPHY PATTERN

I.B.P	-	Ink Blot Pattern
S.S.P	-	Smoke Stack Pattern
RPE C	-	Retinal pigment epithelial changes
PED	-	Pigment epithelial detachment**.**

FUNDUS FLUORESCEIN LEAK PATTERN

FAZ	-	Foveal avascular zone
ITQ	-	Infero temporal quadrant
INQ	-	Inferonasal quadrant
SNQ	-	Superonasal quadrant
STQ	-	Superotemporal quadrant

OPTICAL COHERENCE TOMOGRAPHY (OCT)

PED	-	Pigment epithelial detachment
C.M.T.O.A.E	-	Central Macular thickness of affected eye.
CNVM	-	Choroidal neovascularisation.
S.G	-	Subgroup

MASTER CHART

S. NO.	NAME	AGE	SEX	EYE AFFECTED	D.V	AMSLER'S		PREVIOUS ATTACK			BEST CORRECTED		S.U	FFA PATTERN				FFA LEAK AREAS					OCT PATTERN			sg	V.A		MACULAR	
						C.S	MM/M	S.E	O.E	INTERVAL	R.E	L.E		I.B.P	S.S.P	RPE C.	PED	FAZ	ITQ	INQ	STQ	SNQ	PED	C.M.T.O.A.E	CNVM		4TH WK	12 WK	4TH WK	12 WK
1	GOPI	29	M	RE	YES	YES	NO		YES	5M	6/9P	6/6P	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
2	NARAYANASWAMY	40	M	RE	YES	YES	NO				6/18NIP	6/12PH6/6	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	270	NO	1	6/9NIP	6/6P	197	185
3	KARTHICK	50	M	LE	YES	YES	NO				6/24NIP	6/18NIP	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	334	NO	2	6/18NIP	6/18NIP	317	308
4	BHASKAR	44	M	LE	YES	YES	NO				6/12PH6/6	6/24NIP	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	384	NO	1	6/9NIP	6/9NIP	234	204
5	NARAYANASWAMY	27	M	LE	YES	NO	YES				6/6P	6/18NIP	YES	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	400	NO	2	6/18NIP	6/12NIP	380	350
6	RAJA	23	M	LE	YES	NO	YES		YES	12M	6/9P	6/12NIP	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
7	MEENAKSHI	45	F	RE	YES	YES	NO	YES		6M	6/36NIP	6/24PH6/12	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
8	DASARATHAN	27	M	RE	YES	YES	NO				6/12NIP	6/12NIP	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	287	NO	2	6/12NIP	6/9NIP	275	250
9	KESAVAN	33	M	LE	YES	NO	YES		YES	18M	6/12PH6/9	6/12NIP	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
10	VELMURUGAN	29	M	LE	YES	YES	NO				6/9P	6/18NIP	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	584	NO	1	6/12NIP	6/9NIP	321	208
11	SUNDARAJAN	31	M	LE	YES	YES	NO				6/18PH6/6	6/24NIP	NO	YES	NO	YES	NO	NO	NO	YES	NO	NO	NO	344	NO	2	6/24NIP	6/18NIP	300	280
12	RAVI	23	M	LE	YES	YES	NO		YES	6M	6/9P	6/18NIP	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
13	SEKAR	29	M	RE	YES	NO	YES		YES	5M	6/18NIP	6/12P	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
14	NATARAJAN	35	M	RE	YES	NO	YES				6/18NIP	6/24PH6/9	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	340	NO	1	6/9NIP	6/6P	204	190
15	SRINIVASAN	40	M	RE	YES	NO	YES				6/12NIP	6/24PH6/12	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	278	NO	2	6/12NIP	6/9NIP	240	215
16	VASUDEVAN	47	M	RE	YES	NO	YES				6/36NIP	6/36PH6/18	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	600	NO	1	6/24NIP	6/18NIP	400	250
17	XAVIER	21	M	RE	YES	YES	NO				6/24NIP	6/6P	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	430	NO	2	6/24NIP	6/18NIP	350	295
18	RAMESH	32	M	LE	YES	NO	YES	YES		12M	6/12PH6/6	6/12NIP	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
19	VINOTH	35	M	LE	YES	NO	YES				6/12NIP	6/24NIP	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	367	NO	1	6/12NIP	6/9NIP	210	192
20	VENKATESH	40	M	RE	YES	NO	YES				6/24NIP	6/18PH6/9	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	375	NO	2	6/18NIP	6/12NIP	325	280
21	MANOHAR	43	M	LE	YES	YES	NO				6/9PH6/6	6/36NIP	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	618	NO	1	6/18NIP	6/12NIP	400	280
22	MOORTHY	29	M	RE	YES	YES	NO				6/36NIP	6/6P	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	706	NO	2	6/36NIP	6/24NIP	650	504
23	VELU	36	M	RE	YES	NO	YES		YES	12M	6/12NIP	6/9NIP	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
24	SAIRAM	38	M	LE	YES	YES	NO		YES	6M	6/12PH6/9	6/18NIP	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
25	GOPAL	40	M	LE	YES	YES	NO				6/12PH6/9	6/18NIP	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	376	NO	2	6/12NIP	6/12NIP	326	300
26	KUMARI	40	F	LE	YES	YES	NO				6/18PH6/9	6/24NIP	NO	YES	NO	YES	NO	NO	NO	NO	NO	YES	NO	280	NO	1	6/9NIP	6/6P	198	187
27	RAJENDARAN	48	M	RE	YES	YES	NO		YES	5M	6/36NIP	6/24PH6/9	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
28	MURUGAN	35	M	LE	YES	NO	YES				6/12PH 6/6	6/18NIP	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	380	NO	2	6/18NIP	6/12NIP	340	300
29	SURESH	32	M	RE	YES	YES	NO		YES	6M	6/12NIP	6/12PH6/6	NO	YES	NO	NO	YES	YES	NO	NO	NO	NO	YES		NO					
30	ESWARI	37	F	LE	YES	NO	YES		YES	24M	6/12PH6/6	6/12NIP	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
31	RAJU	39	M	RE	YES	YES	NO				6/18NIP	6/12NIP	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	390	NO	1	6/12PH6/6	6/9PH6/6	210	186
32	LINGUSAMY	45	M	LE	YES	YES	NO				6/24PH6/12	6/24NIP	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	450	NO	1	6//12PH6/9	6/9PH6/6	250	202
33	CHELLAMUTHU	35	M	LE	YES	YES	NO				6/12PH6/6	6/12NIP	NO	NO	YES	YES	NO	NO	NO	NO	YES	NO	NO	275	NO	2	6/12NIP	6/12NIP	260	260
34	RAMESH	27	M	RE	YES	NO	YES				6/36NIP	6/6P	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
35	ANANDAN	29	M	RE	YES	YES	NO				6/18NIP	6/6P	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	376	NO	1	6/12PH6/9	6/9PH6/6	212	197
36	ASHOK	33	M	LE	YES	NO	YES		YES	5M	6/12PH6/6	6/12NIP	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	YES		NO					
37	GOPI	25	M	RE	YES	NO	YES				6/24NIP	6/6P	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	400	NO	1	6/18PH6/12	6/12PH6/6	240	212
38	GANESAN	40	M	LE	YES	YES	NO	YES		36M	6/24PH6/12	6/36NIP	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
39	CHINNAIH	27	M	RE	YES	NO	YES				6/18NIP	6/6P	NO	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	334	NO	1	6/12PH6/9	6/9PH6/6	240	194
40	AMULRAJ	34	M	RE	YES	YES	NO				6/60NIP	6/24PH6/6	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	396	NO	1	6/12NIP	6/12NIP	234	190
41	SUNDARI	31	F	LE	YES	NO	YES	YES		24M	6/12NIP	6/24NIP	YES	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO		NO					
42	KUMAR	27	M	RE	YES	YES	NO				6/18NIP	6/9PH6/6	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	277	NO	2	6/18PH6/12	6/12NIP	260	250
43	RAVI	25	M	LE	YES	YES	NO				6/9PH6/6	6/18NIP	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	YES		NO					
44	KRISHNAMOORTHY	40	M	LE	YES	YES	NO				6/18P	6/18NIP	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
45	THANDAVARAYAN	47	M	RE	YES	NO	YES				6/24NIP	6/36PH6/18	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	624	NO	2	6/36NIP	6/36PH6/24	598	503
46	LOGU	36	M	LE	YES	YES	NO				6/9PH6/6	6/36NIP	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
47	RAMKUMAR	40	M	LE	YES	YES	NO				6/12NIP	6/12NIP	NO	NO	YES	YES	NO	NO	NO	NO	YES	NO	NO	265	NO	1	6/9PH6/6	6/6P	200	180
48	RAJESH	46	M	RE	YES	YES	NO				6/12NIP	6/24PH6/9	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	289	NO	2	6/12NIP	6/12NIP	260	249
49	LAKSHMI	55	F	BE	YES	NO	YES				6/60NIP	6/24NIP	NO	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO		NO					
50	MANI	31	M	LE	YES	YES	NO				6/24PH6/12	6/36NIP	NO	YES	NO	NO	YES	NO	NO	NO	YES	YES	YES	676	NO		6/36NIP	6/24NIP	590	501